## DISSERTATION ON

# "A STUDY ON THE SURGICAL MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION IN ADULTS"

*Dissertation submitted to*

## THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

*In partial fulfillment of the regulations*
*for the award of the degree of*

## M.S. IN GENERAL SURGERY

## BRANCH – I


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## THANJAVUR - 613 004

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# CERTIFICATE

This is to certify that this dissertation entitled "A STUDY ON THE SURGICAL MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION IN ADULTS **"** is the bonafide work of **Dr.AQUINAS. B** in partial fulfilment of the requirements for M.S Branch -I (General Surgery) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in APRIL - 2014 under my guidance and supervision during the academic year march- 2012 to december - 2013.

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## DECLARATION

I, **Dr.AQUINAS. B**, solemnly declare that the dissertation titled **"A STUDY ON THE SURGICAL MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTION ADULTS"** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during March - 2012 to december - 2013 under the guidance and supervision of **Prof. Dr. R. YEGANATHAN M.S. D.A.,** Unit Chief S-VI, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.S. degree (Branch -I) in General Surgery**.

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## ABSTRACT

### BACKGROUND AND OBJECTIVES

Intestinal obstruction remains as one of the most common intra-abdominal pathologies encountered by surgeons whether it is caused by hernia, neoplasm, adhesions or any biochemical disturbances. Intestinal obstruction of the small or large bowel continues to be an important cause of morbidity and mortality. The objective of this study is to analyse the clinical features, treatment and outcome of patients with acute intestinal obstruction along with the cause of obstruction and causes of bowel ischaemia, necrosis and perforation. The following are the objectives of the study:

(1)to study various patterns of presentation, various causes, significance of early recognition, diagnosis and treatment.

(2) to study various influencing factors like age, sex, diet and socio-economic status in the pathogenesis of acute bowel obstruction.

(3)to study morbidity and mortality rates in acute intestinal obstruction.

### METHODS

The materials for the clinical study of bowel obstruction were collected from various Cases getting admitted to various surgical wards. 50 cases of acute

intestinal obstruction have been studied. Patients were belonging to the age groups from 12 years to 85 years, paediatric age group (<12 yrs) is excluded from this study. The case selection criteria was based on history, physical findings, radiological findings and haematological parameters. The study has been divided into Clinical study, Investigations and Treatment. Postoperative Follow-up was done in majority of the patients upto six months after the discharge of patients. The results have been tabulated stressing on following points: age, sex, symptoms, clinical findings, investigations, abnormalities, possible causative factors, operative findings and operative procedure done and complications if any occurred.

## RESULTS

The study group had 50 cases of acute bowel obstruction in the adult age group from 12 years to 85 years. The prevalent age groups are 31-40 and 51-60 age group with around 20% each in the total study. The most common cause of acute intestinal obstruction in the adults in this study series has been post-operative Adhesions (40%) and the next being obstructed Hernia (30%). The clinical features of abdominal pain, vomiting, constipation have been the main symptoms in this study. Abdominal Tenderness, guarding, rigidity, rebound tenderness and shock have been the cardinal features of strangulated obstruction.

The commonest type of obstruction has been due to adhesions or band arising from the previous surgeries. This has constituted to about 40% of the cases of the study group. The second most common type of bowel obstruction is due to obstructed/strangulated external hernia. Salient features had been pain in the groin swelling, acute onset of swelling which is tender, not reducible and without cough impulse. Obstructed hernia constituted about 30% of the total cases studied. Volvulus of the sigmoid colon was 4% in this series. Conservative measures included insertion of ryle's tube and iv fluids but all the cases underwent laparotomy due to failure of the recovery of symptoms. Derotation of the volvulus and sigmoidopexy was done in one case and in one case with vascular compromise, resection and anastamosis was done. Malignancy of the colon was seen in 7 patients constituting 14% of cases. 65% of the malignancies were in the age group of 35-75 years. Of these 2 patients have been managed with Hartman's procedure. One case has been managed with loop transverse colostomy and remaining patients were managed with resection and anastamosis. Most of the deaths occurred those with malignancy.

Although pulmonary tuberculosis is more prevalent in India, due to the use of antitubercular drugs, abdominal tuberculosis is becoming less prevalent. In this study incidence of ileocaecal tuberculosis was 4% and both patients were managed with resection and anastamosis. In this study intussusception causing intestinal

obstruction was 6%. One case was managed with simple reduction and the remaining two patients underwent resection and anastamosis. One case of mesenteric ischaemia was recorded is our study. This patient was managed with resection and anastamosis but patient expired due to septicemia. The complication rate in this study was 18%. Overall mortality of this study was 14%. The results obtained from this study was comparable to various other studies. Malignancy and mesenteric ischemia had more mortality than simple obstruction due to postoperative adhesions. The poor prognosis of the disease was because of late presentation to the hospital with a high incidence of bowel damage with associated faecal peritonitis. The morality in the postoperative period was mostly due to faecal peritonitis, pneumonia and respiratory tract infection.

## INTERPRETATION AND CONCLUSION

Acute intestinal obstruction remains to be an important surgical emergency in the surgical field. Success in the treatment of acute bowel obstruction depends mainly on the early diagnosis and efficient management and treating the pathological effects of the obstruction as much as the treatment of the cause itself. Erect abdomen X-ray is a valuable tool in the diagnosis of acute intestinal obstruction. Postoperative adhesions have been the most common cause to produce bowel obstruction. Clinical, radiological and operative findings when put together can

diagnose the intestinal obstruction. Mortality is still significantly high in acute intestinal obstruction in adults.

**KEY WORDS:** Blood pressure; Computed tomography; Central venous pressure; Superior mesenteric artery; Acute intestinal obstruction; Extra cellular fluid; Intravenous fluids.

# INTRODUCTION

Bowel obstruction remains one of the most common intra-abdominal problems faced by general surgeons in their practice whether caused by hernia, neoplasm, adhesions or related to biochemical disturbances intestinal obstruction of either the small or large bowel continues to be a major cause of morbidity and mortality.[1] They account for 12% to 16% of surgical admissions for acute abdominal complaints. Manifestations of acute intestinal obstruction can range from a fairly good appearance with only slight abdominal discomfort and distension to a state of hypovolemic or septic shock (or both) requiring an emergency operation.

The death due to acute intestinal obstruction is decreasing with better understanding of pathophysiology. Improvement in diagnostic techniques, fluid and electrolytes correction, much potent anti-microbials and knowledge of intensive care. Most of the mortalities occur in elderly individuals who seek late treatment and who are having associated pre-existing diseases like, diabetes mellitus, cardiac diseases or respiratory disease.

Early diagnosis of obstruction skillful operative management, proper technique during surgery and intensive postoperative treatment carries a grateful result.

## OBJECTIVES

1. To study the various ways of presentation, various etiologies, importance of early recognition, diagnosis and management.

2. To study the various influencing factors like age, sex, diet and socio-economic status in the pathogenesis of acute intestinal obstruction.

3. To study the morbidity and mortality rates in acute bowel obstruction.

# REVIEW OF LITERATURE

## HISTORICAL REVIEW

The attempts to treat acute intestinal obstruction goes back centuries. In 6th century, Sushrutha wrote oldest known descriptions of intestinal surgery. Forms of intestinal obstruction like strangulated hernia, intussusception had been known to the ancient Egyptians. Intestinal obstruction had been observed by Hippocrates (460-370 BC). The earliest surgery was performed by Proxogorous (350 BC), who made an enterocutaneous fistula to relieve obstruction.

- Fabricus d'Aquopendente in 12th century AD described a procedure of bowel repair involving end-to-end anastamosis.
- Sanctus in 16th century treated intestinal obstruction by administering metallic mercury to patients.
- John Arderence (1306-1390) was the first person who wrote the book on "Passio Iliaca" (Appendicitis or intestinal obstruction).
- Ambrose Pare (1510-1590) was the first to recognize intestinal obstruction as a pathological entity. For severe patients he used mercury in water, lead bullets smeared with mercury.[3]
- Franco (1561) did the first Surgery on strangulated hernia.

- Kerckring in 1670 described the jejunal valvulae conniventes.
- Johnston in 1938, Harris in 1945, Cantor in 1946 and Gafton Smith in 1952 described many other tubes for gastrointestinal drainage.
- Bishop and Allock (1960) researched the bacteriology of gut above the obstruction.
- Bilgutay (1963) has encouraged the use of faradic current for intestinal paresis following operations.
- The experimental use of Doppler ultrasonography to determine the viability of ischaemic intestine was first described by Wright in 1975.
- Marfuggi and Greenspan in the year 1981 reported 93% accuracy of a fluoroscopic dye injection technique to determine the viability of ischaemic bowel.
- Bookstein in 1982, used angiography for diagnosing and treating small bowel bleeding.
- In 1996, Akgun gained attention in mesosigmoplasty as a definitive operation sigmoid volvulus.[5]
- In 1997, Yaco et al. evaluated the diagnostic procedure for diverticular disease (CT scan, contrast enema, ultrasonography).

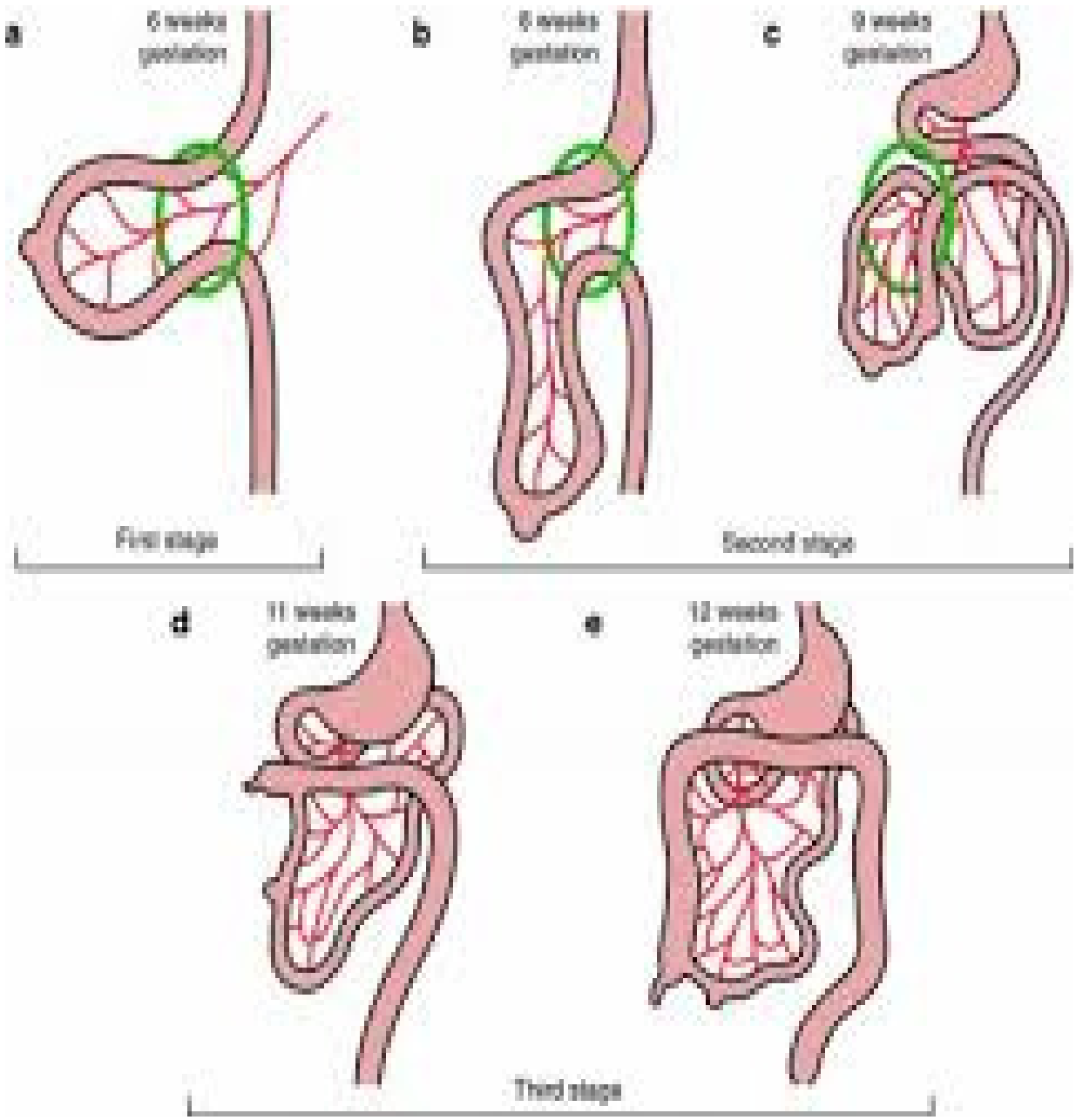
# EMBRYOLOGY OF SMALL INTESTINE

During the early stage of embryogenesis, the primitive bowel is in free communication with rest of the yolk sac. In the cephalic and caudal parts of the human embryo, the primitive gut creates a blind ending tube the foregut and the hindgut and the middle part, the midgut remains temporarily connected to the yolk sac.

In the 5th week embryo, there will be a rapid elongation of the gut and its mesentery resulting in formation of a primary intestinal loop. At its apex, the loop continues to be in open connection with the yolk sac by way of the vitello-intestinal duct. The cephalic part of the loop develops into the distal part of the duodenum, jejunum and part of the ileum. The caudal limb develops into the lower portion of the ileum, the caecum, the appendix, the ascending colon and the proximal two-thirds of the transverse colon. The hindgut develops into distal one-third of transverse colon, the sigmoid, the rectum and also part of anal canal.

## Fixation of the gut

Small and large bowel are suspended from the posterior abdominal wall by Means of a mesentery. After the complete rotation of the gut, the duodenum, the ascending colon, the descending colon and the rectum come to lie in the retroperitoneum by fusion of their mesenteries with the posterior abdominal wall.




**STAGES OF INTESTINAL ROTATION**

There are three errors in the stages of rotation.

1. Non-rotation
2. Reversed rotation
3. Malrotation

**Accessory bands of peritoneum**

Can cause (i) Intestinal obstruction (ii) Kinking (iii) Angulation of bowel. Failure of part of the original membrane to disappear or minor alterations in the development of secondary mesentery may result in accessory peritoneal bands.

These are:

- **Lane's ileal band:** This is a thickened peritoneal band extending from the right iliac fossa to the 5 cm of ileum which on continuous contraction causes kinking of the small bowel and resulting in obstruction.
- **Mesosigmoid membrane** (Lane's first and last band): This is formed by the thickening of peritoneum which extends from the pelvic brim of the left iliac region to the junction of descending and sigmoid colon.
- **Genitomesenteric fold of Douglas:** causes kinking of the appendix causing obstructive appendicitis as it extends from the back of the terminal mesentery to the region of the suspensory ligament of ovary or testis.

# ANATOMY

The small intestine is the longest part of the gastrointestinal tract and it extends from the pyloric orifice of the stomach to the ileocaecal fold. This forms a hollow tube, which is approximately 6-7 m long with narrowing diameter from the beginning to end, it consists of the duodenum, the jejunum and the ileum.[7]

The adult duodenum is 20-25 cm length and the name is coined as duodenum because length is as long as width of 2 fingers. It is shortest, widest and the most fixed part. It does not have any mesentery and is partially covered by peritoneum. Its course presents a remarkable curve somewhat like a horseshoe type, the convexity is being directed towards the right and concavity directed to the left embracing the head of the pancreas. It is divided into four portions. First part (superior portion), Second part (descending portion), third part (horizontal portion) and fourth part (ascending portion).

## Blood supply and nerve supply

Arteries supplying the duodenum are found to arise from the right gastric artery, supraduodenal, right gastroepiploic, the superior and the inferior pancreaticoduodenal arteries.

**Veins:** These end in the splenic, superior mesenteric and portal veins.

**Nerves:** They come from the coeliac plexus.

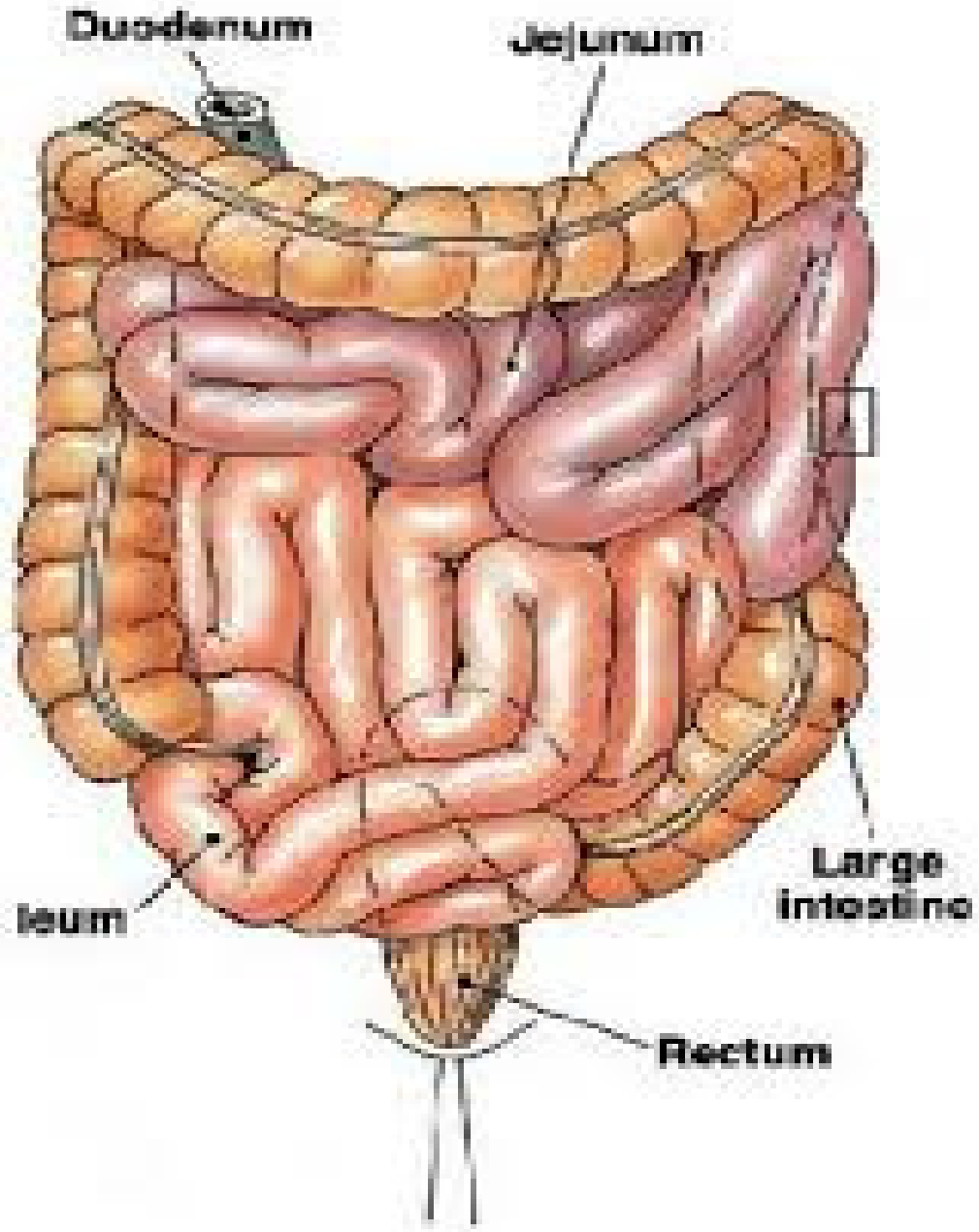
**Lymph nodes:** along inferior and posterior pancreatico duodenal artery.

**Jejunum and ileum**

In small intestine excluding duodenum, upper 2/5 are formed by jejunum and lower 3/5 are ileum. The rest of the small intestine extends from the duodeno-jejunal flexure to the ileo-caecal valve, ending at the junction of the caecum and ascending colon. It is totally covered by the peritoneum and it is arranged in a series of coils attached to the posterior abdominal wall by the mesentery. The jejunal loops are characteristically situated in the upper abdomen to the left of the midline, whereas the ileal loops tends to lie in the lower right part of the abdomen and pelvis. This distribution can be reversed during paralytic ileus or small bowel obstruction due to rotation around the mesentry attachment following bowel distension.

The wall of jejunum and ileum is composed of serosa of visceral peritoneum, muscular of longitudinal and circular smooth muscle fibres and a mucosa of connective tissue, smooth muscle and epithelium.

# Regions of the Small Intestine



**Blood supply**

Blood supply is by the superior mesenteric artery which is a branch of abdominal aorta, the branches of which, reaching the mesenteric border, extend between the serosal and muscular layers. After this, numerous branches traverse the muscle, supplying it and thus forming an intricate submucosal plexus from which many minute vessels pass to glands and villi. The superior mesenteric veins correspondingly follow the arteries.

**Nerve supply**

Nerve supply is by vagi and thoracic splanchnic nerves through the celiac ganglia and the superior mesenteric plexus.

**Large intestine**

It is about 150 cm in length, it extends from the terminal ileum to the anus. Its function is mainly absorption of fluids and solutes and it differs in its structure, size and arrangement from the small bowel in the following ways:

- It is for the most part fixed in position.
- Its longitudinal muscle, though being a complete layer, is concentrated into three longitudinal taeniae coli.

- The colonic wall is puckered into sacculations (haustrations) and appendices epiploicae by the taeniae.
- It has a great diameter.

The divisions are the caecum, colon proper and the rectum.

**Caecum**

The caecum is like a blind pouch that lies in the right iliac fossa, with its average axial length being about 6 cm and its breadth is about 7.5 cm. this continues proximally with the distal ileum and distally it continues with the ascending colon and it is related posteriorly to iliopsoas muscle and the femoral nerve, anteriorly related to the abdominal wall, greater omentum and the loops of ileum. Almost the entire posterior part of the caecum is attached to the abdominal wall, but in some cases it is wholly unattached.

**Ileocaecal valve**

The ileum opens onto the posteromedial aspects of the caeco-colic junction and two flaps which form a projection into the lumen of the colon. The valve is actually closed by sympathetic tone. It is mechanically closed during the distensions of caecum and this prevents the reflux of contents of the caecum into the ileum and thus regulates the flow of ileal contents.

## Colon

The colon is conveniently sub-divided in four parts: (1) Ascending, (2) Transverse, (3) Descending and (4) Sigmoid colon.

### Ascending colon

It is usually fused with the posterior body wall and is covered by peritoneum anteriorly. It constitutes about 15 cm in length and is narrower than the caecum. It ascends onto the inferior surface of the right lobe of the liver, on which it causes a shallow depression. Here, it turns abruptly forwards and to the left forming right colic flexure.

### Hepatic flexure

Anteriorly it is covered by the peritoneum, but posteriorly it is not covered by peritoneum and It lies in direct contact with renal fascia. It is related posteriorly to the infero-lateral part of the anterior surface of the right kidney above, and anterolaterally lies the right lobe of the liver, anteromedially are the descending part of the duodenum and the fundus of the gallbladder.

### Transverse colon

It extends from the hepatic flexure to the left colic flexure measuring 50 cm. The transverse colon, which unlike ascending and descending colon has a mesentery

that had secondarily fused with posterior wall of the omental bursa. The transverse colon hangs in either U or V shaped curve. Above the transverse colon lies the liver and gallbladder, the greater gastric curvature and the lateral end of spleen, below lies the small intestine, in front are the posterior layers of the greater omentum and behind are the descending part of the duodenum, the pancreatic head, the upper end of the mesentery, duodenojejunal flexure and the loops of the jejunum and ileum. The transverse colon sometimes may be interpositioned between liver and diaphragm (Chilaiditi syndrome).

**Splenic flexure**

This is the junction of the transverse and the descending colon in the left hypochondriac region. It is closely related to the lower part of the spleen and pancreatic tail above and medially with the anterior of the left kidney. It is attached to diaphragm by means of phrenico-colic ligament, which lies below the antero-lateral pole of the spleen. It lies more superiorly and more posteriorly than the hepatic flexure at the level of 10th and $11^{th}$ ribs.

**Descending colon**

It is about 25 cm in length and it extends from the splenic flexure to pelvic brim, and in the whole of its course, it is plastered to the posterior abdominal wall by peritoneum (like ascending colon). The descending colon is much smaller in

caliber more deeply placed and more frequently covered posteriorly by peritoneum. The descending colon lies on the lumbar and iliac fascia. It ends at the pelvic brim about 5 cm above the inguinal ligament.

**Sigmoid colon**

It is about 40 cm in length. Sigmoid colon extends from the descending colon at the pelvic brim to the commencement of rectum in front of the third piece of the sacrum. The sigmoid mesocolon has an inverted 'V' attachment to the posterior abdominal wall.

**Blood supply**

Blood supply is by the branches from the superior mesenteric artery and inferior mesenteric artery. Superior mesenteric artery supplies upto the junction of middle 1/3$^{rd}$ of transverse colon and colon beyond this is supplied by the inferior mesenteric artery.

**Nerve supply**

Sympathetic nerve supply to the midgut from coeliac ganglion (T1-L1). Parasympathetic supply is from vagus through the coeliac plexus.

Hindgut portion receives sympathetic nerve supply from the lumbar sympathetic chain from the L1-L2 and parasympathetic supply from the pelvic splanchnic nerves.

**Rectum**

The rectum is about 12 cm long and it is continuous with the sigmoid colon at S3. The human rectum follows the posterior concavity of sacrum and shows three lateral curves or flexures that are most prominent when the viscus is distended, upper and lower curves are convex to the right and a middle curve is convex to the left, the lowest part is slightly dilated as the rectal ampulla. It ends 2-3 cm in front and below the tip of the coccyx, turning abruptly downwards and backwards through the levator ani muscle to become the anal canal 4 cm from the anal verge.

**Blood supply**

Blood supply is mainly from the superior rectal artery, with contributions from the middle, inferior rectal and median sacral vessels. Veins correspond to the arteries, but they anastamose freely with one another, forming an internal rectal plexus in the submucosa and external rectal plexus outside the muscular wall.

**Nerve supply**

The sympathetic nerve supply is derived by branches from the hypogastric plexus. The parasympathetic supply is from S2 and S3 by the pelvic splanchnic nerves.

**Lymphatic drainage of colon**

Lymph from the colon passes through 4 sets of lymph nodes: (a) Epicolic lymph nodes, lying on the wall of the colon, (b) the Paracolic nodes lying on the medial side of ascending, descending and mesocolic border of transverse and sigmoid colon, (c) Intermediate nodes along the main branches of the vessels, (d) Terminal nodes at the origin of SMA and IMA, finally drains into para-aortic nodes.

**PHYSIOLOGY**

The gastrointestinal system consists of the gastrointestinal tract and the associated glandular organs that produce the gastro-intestinal secretions. The major physiological functions of the gastrointestinal system are to digest food stuffs and to absorb nutrient molecules into the blood stream. Mainly the small intestine and large intestine carry out these functions by motility, secretion, digestion and absorption.

Motility of the bowel refers to the movements that mix and circulate the gastrointestinal contents and propel them along the length of the gatrointestinal tract. The contents are usually propelled in the antegrade (forward) direction.

Secretion – refers to the processes by which glands in the GIT associated with the small intestine and large intestine release water and other substances into the lumen.

Digestion – defined as the processes by which the food and large molecules are chemically degraded to produce smaller molecules that can be easily absorbed along the wall of the bowel.

Absorption refers to the processes by which nutrients are absorbed by cells that live in the intestine and enter the circulation.

**Properties of succus entericus**

Volume – 180 ml/day

Reaction – Alkaline

pH – 8.3

## Functions of Succus Entericus[9]

1. Digestive function – The enzymes of succus entericus act on the partially digested food and convert them into the final digestive products.
2. Protective function – The mucus present in the succus entericus helps in protecting the intestinal wall from the acid chime, which enters into the intestine from stomach paneth cells secrete defensins which are the antimicrobial peptides.
3. Activator function – The enzyme enterokinase present in intestinal juice activates trypsinogen into trypsin.
4. Haemopoietic function – Intrinsic factor of castle present in the intestine plays important role in erythropoiesis.
5. Hydrolytic process – Intestinal juice helps in all the enzymatic reactions of digestion.

## Functions of small intestine

1. Mechanical function
2. Secretory function
3. Hormonal function
4. Digestive function
5. Activator function

6. Hemopoietic function
7. Hydrolytic function
8. Absorptive function

**Large Intestine**

**Secretions**

Large intestine juice

Water 99.5%

Solids 0.5%

Organic substances

1. Albumin

2. Globulin

**Functions of large intestine**

1. Absorptive function – absorption of various substances such as water, electrolytes, organic substances like glucose, alcohol, drugs like anaesthetic agents, sedatives and steroids.

1. Excretory function
2. Secretory function
3. Synthetic function – synthesis of folic acid, vitamin B12 and vitamin K

**Movements of small intestine**

The movements of small intestine is essential for the mixing of chime with digestive juices, propulsion of food and for its absorption.

**Four stages of movements take place in the small intestine.**

1. Mixing movements
    a. Segmentation movements
    b. Pendular movements
2. Propulsive movements
    a. Peristaltic movements
    b. Peristaltic rush
3. Peristalsis in fasting – Migrating motor complex
4. Movements of the villi

**Movements of large intestine**

- Segmental contractions
- Mass peristaltic movements.

**Intestinal bacteria**

The bacteria in the gastrointestinal tract can be divided into three types.

1. Some are pathogens that cause disease.
2. Others are symbionts that benefit the host and vice versa, and most of them are commensals.

## PATHOPHYSIOLOGY OF BOWEL OBSTRUCTION

Management of acute bowel obstruction depends largely on early recognition, skillful management and appreciation of the importance of treatment of the pathological effects of the obstruction just as much as the etiology itself.

If detected early, the prognosis will be excellent after relief of obstruction but in late cases, where there will be vascular compromise due to obstruction, where relief of obstruction is not enough, rather it calls for many other surgical procedures like resection, anastamosis, etc.

### .Pathophysiological changes in acute bowel obstruction

### 1. Intestinal distension

Although being a constant feature of bowel obstruction, the mechanism underlying the intestinal distension has not been described completely. Most of the gas distending the small bowel in early phases of bowel obstruction accumulates

from swallowed air. Other sources include: fermentation, production of carbon dioxide by interaction of acid from stomach and bicarbonates in pancreatic and biliary secretions, and the diffusion of oxygen and carbon dioxide from the blood. Following dilatation and inflammation, activated neutrophils and macrophages accumulate in the bowel wall because increased blood flow to the gut. These release reactive proteolytic enzymes, cytokines, and other locally active substances which either inhibit or damage the secretory and the motor processes of the gut. The nitric oxide produced during the process causes smooth muscle relaxation, further aggravating the bowel distension and inhibiting gut contractility. The normal intraluminal pressure of 2 to 4 cm of water increases to 8 to ‘to cm of water in case of intestinal obstruction, which may reach upto 30 to 60 cm of water in close loop obstruction. The reactive oxygen radicals produced during these changes not only affect gut motility but also gut permeability.

During the first 12 hours of obstruction, water and electrolytes accumulate within the lumen secondary to a decrease in absorption. By 24 hours, accumulation occurs more rapidly because of a further decrease in absorption and in addition to the increase in intestinal secretion secondary to mucosal injury and increased permeability. Although the role of neural or systemic humoral/hormonal mechanisms in aggravating the distension remains likely, it has been poorly investigated. This decrease in the absorptive capacity of the gut associated with an

increase in intraluminal secretion leads to excessive fluid losses which can further lead to dehydration. Although the intestinal wall distal to the obstruction maintains its normal function, the inability of the intra-luminal content to reach the unobstructed gut further compounds the dehydration.

**2. Intestinal Motility**

Intestinal motility, absorption and secretion are also altered after intestinal obstruction but the point at which the normal bacterial barrier function of the viable gut fails after intestinal obstruction is still unclear.[10]

In the early phase of intestinal obstruction, intestinal contractility increases in an attempt to propel intraluminal contents past the site of obstruction. Later, it diminishes secondary to intestinal wall hypoxia and exaggerated intramural inflammation; However, the exact mechanisms behind this have not been completely elucidated. Some investigators have suggested that the alterations in bowel motility are secondary to a disruption of the normal autonomic, parasympathetic and sympathetic & splanchnic innervation.

**3. Circulatory Changes**

Ischaemia of the intestinal wall occurs by several different mechanisms. Extrinsic compression of the mesentery by the presence of adhesions, fibrosis, mass, twisting or a hernia defect, extrinsic pressure on a segment of bowel wall

(e.g., a fibrous band), or progressive distension in the setting of a closed-loop obstruction can all cause vascular compromise or strangulation. The consequences of the vascular compromise are more disastrous in large intestinal obstruction, as in nearly a third of people ileocaecal valve is competent,11a which can functionally lead to a closed-loop obstruction between the competent ileocaecal valve and the site of obstruction in the large intestine.

Progressive distension of the bowel lumen with a concomitant rise in intraluminal pressure leads to increased transmural pressure on capillary blood flow within the bowel wall. In the simple (non-closed loop) obstruction, this occurs rarely, as the obstructed distended intestine can decompress proximally. Severe intestinal distention, however, is self- perpectuating and progressive, intensifying the peristaltic and secretory abnormalities and increasing the risks of dehydration and progression to strangulating obstruction. Strangulation is obstruction with compromised blood supply; it occurs in nearly 25% of patients with small bowel obstructions. It is usually associated with obstructed hernia, volvulus, and intussusception. Strangulating obstruction may progress to infarction and gangrene of the bowel in as little as 6 hours. Venous obstruction usually occurs first, followed by arterial occlusion, resulting in rapid ischemia of the bowel wall. The ischemic bowel becomes edematous and infarcted, leading to gangrene and perforation. It is more common in case of caecum and ascending colon where the

luminal diameter is greatest and (by Laplace's law) the wall tension (and ischemia.) is also maximum. This makes large bowel obstruction a more surgical emergency than small bowel obstruction. With strangulation, there also can be blood loss into the infarcted bowel, which together with the preexistent fluid loss can lead to further hemodynamic instability, exacerbating the already compromised blood flow to the intestinal wall.

**4. Microbiological changes and Bacterial Translocation**

The upper small bowel contains gram-positive facultative organisms in small concentrations, usually <1000000 colonies/mL. More distally, the bacterial count rises in concentration to about 108 colonies/mL in the distal ileum, with flora changing mainly to coliforms and anaerobes. In the presence of obstruction, bacteria can proliferate rapidly proximal to the obstruction in direct proportion to the duration of intestinal obstruction, reaching a plateau of 109-1010 colonies/mL after 12-48 hours of obstruction. The bowel distal to the obstruction tends to retain its usual bacterial flora until paralytic ileus sets in, following which there is generalized bacterial proliferation. Toxins produced by these bacteria disrupt the mechanical integrity of the intestinal mucosa. Once the gut mucosal barrier is lost, translocation of bacteria occurs as the luminal bacteria invade the submucosa and enter the systemic circulation via the portal and lymphatic systems.

Due to these alterations in resident microbial flora, the risk of infective complications in bowel obstruction has increased markedly, especially if bowel resection is required or if any inadvertent enterotomy is made with intraperitoneal spillage of "obstructed" intestinal contents. With strangulation, there is systemic entry of bacterial products, the activation of immunocompetent cells, release of cytokines, and increased formation of reactive oxygen intermediates, this eventually leads to systemic inflammatory response syndrome and multiple organ dysfunction syndrome.

**PATHOLOGY**

Distension occurs proximal to the obstruction and it starts immediately after the obstruction begins. Causes of bowel distension are: fluid, gas and intestinal toxins.

**(a) Fluid:** made up by the various digestive juices about 8 litres/day.

In obstructive pathology, there will be increased enteric pressure leading to edema, shortening and clubbing of the intestinal villi leading to disturbed absorption. Also there will be a depletion of water and electrolytes due to vomiting, defective absorption, sequestration within the lumen of bowel.

**(b) Gas:** Consists of swallowed atmospheric air, diffused air from blood into the intestinal lumen 22% and the products of enzymatic digestion and bacterial

activity. When the $O_2$ and $CO_2$ has been absorbed into the blood stream, the resulting mixture is made up of $N_2$ (90%) and $H_2S$.

**(c) Intestinal toxins:** In unrelieved strangulation, toxic substances tend to appear in the peritoneal cavity only when the viability of the intestinal wall is affected. When the obstruction is relieved, these toxic products may pass on to the bowel where absorption occurs. It is probable that the substances involved are mainly endotoxins of Gram negative bacilli.

The first effect of strangulation is compression of the veins so as to cause ischaemia. When the venous return is totally occluded, the colour of the intestine turns from purple colour to black. Due to raised edema at the point of obstruction, there will be rupture of capillaries with haemorrhagic infiltration. Thrombosis in intramural and mesenteric veins augments the ischaemia. Mucosal necrosis first appear and spread towards the serosa causing wet gangrene of the bowel.

Increasing venous and arterial occlusion causes extravasation of blood under the serosa and effusion into the lumen of the intestine. In the case of strangulated external hernia , only a small segment of the bowel is involved, the blood that is sequestrated is minimal but when a large coil of the gut becomes strangulated, the blood loss is quite significant to make the patient hypovolemic and in severe cases this leads to death of the patient.

In case of obstruction due to closed loop as seen in the case of carcinomatous stricture of the colon. Proximally the competent ileocaecal valve prevents the regurgitation of contents whereas distally the colon is obstructed by the neoplasm. This leads to high pressure inside the caecum. If obstruction is not relieved, due to compression of the blood vessel in wall, ulceration, gangrene and eventually perforation of the caecum will occur.

**Adynamic obstruction**

Adynamic obstruction is a condition when there is failure of neuro-muscular mechanism i.e. the Auerbach's and Meissener's plexuses resulting in atony of the intestine, causing loss of peristalsis and accompanied by abdominal distension.

The factors that leads to paralytic ileus are:

Humoral

Neural

Drugs

Postoperative

Metabolic

## CLASSIFICATION OF INTESTINAL OBSTRUCTION

Acute intestinal obstruction is most commonly a disorder of small intestine and accounts for approximately 20% of all the surgical admissions.

Intestinal obstruction can be classified into two types.

1. Dynamic obstruction
2. Adynamic obstruction

**DYNAMIC OBSTRUCTION:** Where peristalsis is working against a mechanical obstruction.

Irrespective of the aetiology or acuteness of the onset, in dynamic obstruction the proximal intestine dilates and develops an altered motility. Below the obstruction the intestine exhibits normal peristalsis and absorption until it becomes emptied at which point it tends to contract and becomes immobile.[11]

The causes of intestinal obstruction can be :[12]

**Intraluminal**

- Intussusception
- Bezoar
- Foreign bodies
- Gallstones

- Mucosal tumours

**Intramural**

- Stricture
- Malignancy: Carcinoid, Lymphoma, Leiomyosarcoma
- Inflammation: Crohn's disease, Tuberculosis
- Haematoma
- Endometriosis

**Extramural**

- Bands/adhesions
- Hernia: External – Inguinal, Femoral, Incisional, Obturator hernias

  Internal – Paraduodenal, Epiploic foramen, Diaphragmatic, Transmesenteric hernias.

- Tumours: Peritoneal metastasis, Desmoid tumour.
- Abscess: Diverticulitis, Pelvis inflammatory disease, Crohn's disease

**ADYNAMIC OBSTRUCTION:** This can occur in two forms:

a. Peristalsis can be absent e.g.: Paralytic ileus
b. Peristalsis may be present but in a non-propulsive form e.g.: (1) Mesenteric vascular occlusion (2) Pseudo-obstruction.

## CLINICAL FEATURES

Cardinal features of acute intestinal obstruction are:

1. Abdominal pain.
2. Vomiting
3. Abdominal distension.
4. Constipation

### 1. Pain Abdomen.

Abdominal pain is the usually first symptom. The onset may be insidious or abrupt in simple obstruction, but with strangulation the onset is sudden and severe. The pain will diffuse, poorly localized and is felt across the upper abdomen in case of high obstruction, at the level of the umbilicus in low ileal obstruction, in the lower abdomen in colonic obstruction and in the perineum in rectosigmoid obstruction.

### 2. Vomiting

Vomiting is the next most common symptom. Being a constant symptom, the early vomiting is reflex in nature followed by quiescent period, before real vomiting due to obstruction begins. This quiescent period will be of shorter duration in high-level obstruction and longer in lower small intestinal obstruction. As acute intestinal obstruction progresses, the character of the vomitus changes.

Initially it contains partly digested food, followed by bilious vomiting. Finally it becomes faeculent.

### 3. Distension

In the early cases of obstruction of the small intestine, abdominal distension is often slight or even may be absent. When the proximal jejunum is obstructed, the stomach gets distended with gas and the accumulated secretions, so that the epigastric region may, in later stages become more prominent and tense. When the ileum is involved, the central part of the abdomen is moderately blown out and when the distal colon is blocked, there will be considerable universal distension of abdomen, with well-marked bulging in the region of flanks. Visible peristalsis can be present.

### 4. Constipation

In complete obstruction, after the contents of the intestine below the obstruction have been evacuated, there will be constipation and usually neither the faeces nor flatus is passed i.e. absolute constipation.

The rule that constipation is present in intestinal obstruction does not apply in cases with Richter's hernia, gall stone obstruction, mesenteric vascular occlusion.

## History and physical examination

A detailed history and physical examination usually helps in the diagnosis and management of intestinal obstruction. In simple mechanical obstruction, there will be very few abdominal signs. Whereas with strangulated obstruction, patient will be toxic, tachycardiac and hypotension will be there. Any past history of abdominal surgery, acute cholecystitis, appendicitis or any other intra-abdominal infections, suggests adhesion to be the cause of obstruction. Hernia of long duration gives rise to strangulation. There may be the following history:

- Alternate diarrhoea and constipation with weight loss suggests tuberculosis and malignancies.
- Recent onset constipation suggests malignancy in elderly people.

## Physical examination

**Skin turgor:** can be lost due to dehydration, may be cold and calmy.

**Tongue:** which may become dry and coated due to dehydration.

**Nail and sclera:** Anaemia or jaundice may be evident.

Rapid low volume pulse, low blood pressure, cold extremities, anxious look and increased respiratory rate are indicative of shock and septicaemia.

**Examination of abdomen**

- **Inspection:** On inspection previous surgical scar indicates adhesions or cancer. In early stage visible peristalsis can be seen. All hernial orifices has to be examined.
- **Type of abdominal distension:**
- **Type of peristalsis:**

**Palpation**

- Abdomen must be examined for presence of any palpable mass, localized tenderness, rebound tenderness which may be suggestive of strangulation. In peritonitis there will be a generalized rigidity and tenderness. During pain heaped up coils of intestine or prominent distended coils can be seen.
- **Auscultation:** In simple mechanical obstruction, sounds will become loud, high pitched and metallic. In late stages bowel sounds will be absent due to paralysis of bowel musculature. Bowel sounds can be absent in strangulation and ileus or low-pitched tingling sounds will be heard due to movements of fluid from one coil to another.
- **Rectal examination:** must be performed in all cases of obstruction, may reveal faecal impaction, mass, red currant bleeding in intussusception. A palpable pelvic mass or bulge because of collection in the Pouch of Douglas

may be present. Ballooning of the rectum which usually occurs in the intestinal obstruction may be due to obstruction to nerves which causes sympathetic paralysis.

## CLINICAL FINDINGS IN SMALL BOWEL OBSTRUCTION

| Features | Proximal/High obstruction | Distal/low obstruction |
|---|---|---|
| Onset of symptoms | Sudden | Gradual |
| Pain | Epigastric, intense, colicky usually relieved by vomiting | Periumbilical, colicky |
| Vomiting | Early, bilious, voluminous, frequent | Later, infrequent feculent |
| Tenderness | Epigastric / periumbilical mild unless strangulated | Diffuse and progressive |
| Distension | Absent | Diffuse and progressive |
| Obstipation | Absent or mild | Mild to moderate |
| Radiologic findings | Distended proximal small bowel loops or gasless | Diffusely distended small bowel loops, air fluid levels |

## LABORATORY INVESTIGATIONS

### (i) Haematological tests

A complete blood count, packed cell volume, serum electrolyte determination and blood urea level estimation should be done. Leucocytosis is suggestive of strangulation, extremely high counts are mainly suggestive of mesenteric thrombosis.

### (ii) Urine examination

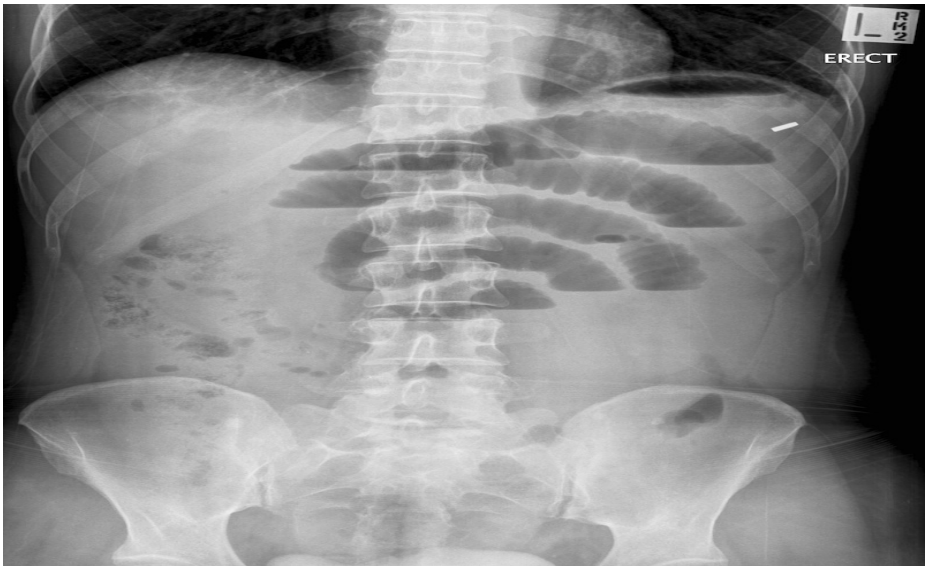
Specific gravity of the urine will give a rough idea of the degree of dehydration in case of intestinal obstruction.

### (iii) Diagnostic aspiration

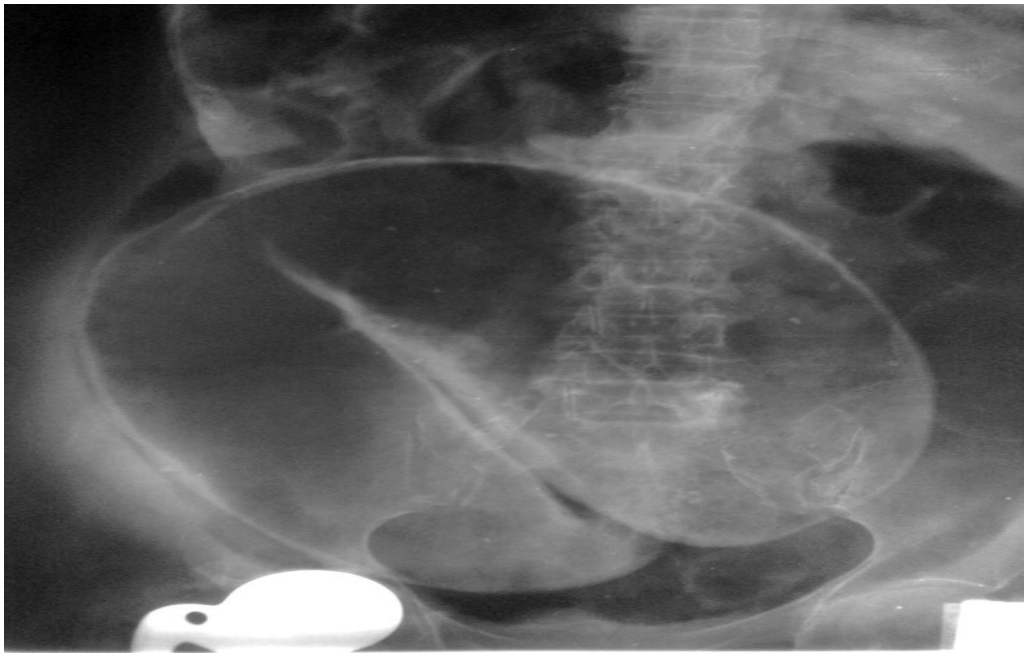
It is very important in the distinction between simple and strangulated obstruction. Aspiration of peritoneal cavity with a fine needle and withdrawal of blood stained fluid is diagnostic of strangulation.

### X-ray diagnosis

The finding in erect abdominal X-ray for small intestinal obstruction is the triad of dilated small bowel loops (>3 cm in diameter), air fluid levels on upright films and paucity of air in the colon.[15]




**MULTIPLE AIR-FLUID LEVELS**



**COFFEE BEAN APPEARANC**

**Gas shadows:** When the jejunum, ileum or the colon are distended with gas, each structure has significant radiological images. Jejunum is characterised by 'valvulae conniventes' that pass from the anti-mesenteric to the mesenteric border in the regular fashion. Ileum radiography is described by Wangensteen as being 'characterless'. Large intestine reveals haustral markings which unlike the valvulae conniventes are spaced irregularly and they do not completely traverse the circumference of the bowel and gas shadows of large intestine will be located peripherally.

**Fluid levels:** In adults, two inconstant fluid levels are regarded as physiological, one at the duodenal cap and the other in the terminal ileum. In obstruction, fluid levels tend to appear later than the gas shadows; the number of fluid level is proportional to the degree of obstruction and to its location in the small intestine.

In upper small intestinal obstruction, fluid levels will be in left upper quadrant and they will be few in number, multiple fluid level seen all over the abdomen in case of lower small bowel obstruction. The presence of gas within the wall of the bowel is highly significant sign of bowel necrosis, which was demonstrated by Schorr in 1963.

Volvulus of sigmoid colon shows greatly distended sigmoid loop filling the whole of the abdomen till the diaphragm with the "Bent inner tube sign". Millin

and Righler pointed out that the “coffee bean” sign in pathognomonic sign of caecal volvulus.

**Barium enema:** In intussusception, barium is seen like a ‘claw’ around a negative shadow of intussusceptions, whereas in sigmoid volvulus, barium column ends at the level of the distal sigmoid in a characteristic Twisted Bird’s Beak deformity.

**Computerized tomography (CT)**

CT demonstrates the cause of intestinal obstruction.

Computerized tomography is very much useful in establishing the site, level and the cause of obstruction and in displaying signs of threatened intestinal viability. CT scan is most valuable when there are systemic signs indicative of infarction, an associated palpable abdominal mass. In these cases CT scan may confirm the presumptive diagnosis or reveal other causes mimicking obstruction such as appendicitis or diverticulitis. In strangulated obstruction, target sign or pneumatosis intestinalis and haemorrhage in the mesentery can be seen.

**TREATMENT OF ACUTE INTESTINAL OBSTRUCTION**

With few exceptions, an urgent intervention is needed in a case of intestinal obstruction. Eventhough it is difficult to differentiate a simple and strangulated obstruction, the assessment of the patient is done by taking a detailed history and

clinical examination. Investigations are done to find out whether the obstruction is mechanical or adynamic and to make out the level of obstruction.

The treatment must be planned according to the above assessment which includes both supportive management and surgical management. There are four important measures in the management of obstruction.

**Conservative management**

Cases with a partial intestinal obstruction can be treated conservatively with resuscitation and tube decompression alone. Regression of the symptoms and discharge without the need of surgery have been reported in 60-85% of patients with a partial obstruction.[16] Simple obstruction caused by post-operative early adhesions or kinking may resolve spontaneously with the institution of conservative management and it is indicated in following situations.

- Post-operative early adhesions
- Paralytic ileus of non-paralytic origin
- Inflammatory condition which cause obstruction
- Obstruction because of worm impaction

The initial conservative management is used in the above said conditions. This facilitates spontaneous relief of obstruction and avoids more adhesion formation due to surgery. The decision to operate depends largely on the underlying cause,

clinician involved and general status of the patient. The conservative management includes.

- GI decompression
- Fluid and electrolyte therapy.
- Antibiotics.

**Surgical Management**

With regard to the timing of the operation, all patients should be operated on promptly after volume resuscitation if there is any evidence or suspicion that bowel is ischaemic.[17] Early surgery indicated in (1) obstructed and strangulated hernia, (2) internal intestinal strangulated obstruction (3) acute obstruction, The classical clinical saying that the sun should not set and rise in a case of unrelieved intestinal obstruction is sound and it should be followed.

**Laparotomy**

In patients with small intestinal obstruction, who have not had any previous abdominal surgery or in those with clinical evidence of ischaemia, a laparotomy is essential.[18]

When the cause of intestinal obstruction lies within the abdomen and but its site is doubtful, right paramedian incision is advised, if left sided large bowel

obstruction is defined left mid or lower paramedian incision may be preferred, abdominal cavity is inspected which reveals the underlying pathology. Haemorrhagic fluid suggests strangulation; clear straw-coloured fluid denotes simple obstruction. The operative assessment is directed at:

- Site of obstruction
- Viability of the bowel
- The nature of obstruction

The type of surgical procedure required will depend on the nature of the cause, following relief of obstruction the viability of the involved segment of bowel should be carefully assessed. In case of a viable bowel, peritoneum will be shiny, mesentery bleeds on prick whereas with a nonviable bowel, peritoneum is lusterless, mesentry does not bleed on prick.

**Difference between viable and non-viable bowel**

| Intestine | Viable | Non-viable |
|---|---|---|
| Circulation | Dark colour becomes lighter, mesentery bleed on prick | Dark colour remains mesentery does not bleed on prick |

| | | |
|---|---|---|
| Peritoneum | Shiny | Dull and lusterless |
| Intestinal musculature | Firm, Pressure rings may or may not disappear, peristaltic movements may be observed | Flabby, thin and friable pressure rings persist, no peristaltic movements are observed |

Doppler ultra-sonography can also help to establish the circulation in the mesenteric vasculature (a most accurate modality of testing viability). If viability of the bowel is in doubt, it should be placed in warm moist pads for ten minutes along with administration of 100% oxygen in the anaesthetic gas. Then after ten minutes it has to be re-examined, in doubtful cases resection has to be done.

**Principles of large bowel obstruction**

As most of the large intestinal obstruction are due to malignancy, volvulus or secondary to adhesive bands, which more commonly occur, in elderly patient.

When the lesion is operable and found in the caecum, ascending colon or proximal transverse colon, a right hemicolectomy should be performed; if lesion is fixed, a proximal stoma (colostomy. or ileostomy if ileocaecal valve is incompetent) or an ileo-transverse colonic bypass has to be considered, whereas

obstructing lesions of the splenic flexure (malignant) should be treated by an extended right hemicolectomy. If one stage resection and anastomosis is not feasible a covering colostomy to protect the site of anastomosis is safer, where the distal segment could not be brought to the surface a proximal stoma and the distal end can be closed and returned to abdomen (Hartman's procedure) or both the ends may be brought outside, proximal as stoma and distal as mucus fistula, followed by second stage colorectal anastomosis which can be planned when the patient is fit. In very old or cachectic patients when an obstructing carcinoma of rectum which is fixed, left iliac colostomy is the best site for the placement of a permanent artificial anus. In rare circumstances, or if caecal perforation is strongly suspected, we should wait for some time for improvement of the patient's condition and later relief of obstruction can be done by doing an emergency caecostomy through a small incision in the right iliac fossa.

**INTESTINAL OBSTRUCTION BY ADHESIONS AND BAND**

It is the most common cause of intestinal obstruction in the developing countries. The pathology lies with peritoneal irritation resulting in local outpouring of fibrin which produces adhesions between the opposed peritoneal surfaces. These fibrinous adhesions may become vascularised and become a mature fibrous tissue, infection being an important cause. Also foreign materials like the silk thread, barium sulphate, talc, results in fibrous formation.

These commonly occurs following laparotomies. Once adhesions have developed, progression to obstruction is very much inevitable in a significant proportion.[19] Ileum is the most common segment to be obstructed due to adhesions. After abdominal surgeries, about 5% of the patients subsequently develop obstruction due to adhesions, whereas operation in the colon carry a high incidence of obstruction, about 1/5th of cases. About 20% of the obstruction occurs within the first year after laparotomy and most of these occur during the first few weeks after surgery and are termed as early postoperative obstructions and most get resolved by conservative treatment.[20]

**Treatment**

The treatment for adhesions is the same as the general principles of management often is curative in early type of adhesions, but conservative treatment should not be prolonged beyond 48-72 hours and should not be continued if symptoms and signs are progressive even after initial resuscitation. When laparotomy is done multiple adhesiolysis should be done.

**Recurrent intestinal obstruction due to adhesion**

Adhesions are a major cause of late morbidity. Approximately 10% of all patients who had laparotomy for adhesive obstruction will require a further

operation at a later date for the same issue. A further 10% may require a third operation for adhesive obstruction.

There are often chances of recurrent obstruction after the first adhesiolysis. The following procedures may be considered for recurrent adhesive obstruction

- Intestinal intubation
- Charles- Phillips transmesenteric plication
- Noble plication
- Repeat adhesiolysis (enterolysis)

**Internal hernias**

When a segment of the small intestine becomes herniated into one of the retroperitoneal Fossae, it is termed as internal hernias. This can occur in following sites:

- Supra-vesical hernia
- Foramen of Winslow
- Diaphragmatic hernia: Acquired/Congenital.
- Caecal or appendiceal: Retroperitoneal fossae superior or retrocaecal
- A hole in the mesentery or mesocolon and defects present in the broad ligament.

- Paraduodenal fossae: Right/left paraduodenal fossae.

Internal herniation in the absence of adhesions is quite uncommon and a preoperative diagnosis is unusual. The standard treatment for a hernia is to release the constricting agent by divison.[11]

Internal herniations are treated by laparotomy and the release of constriction ring. The distended loop first needs to be decompressed and then reduced. Unviable segment of the bowel has to be resected and anastomosed.

**INTUSSUSCEPTION**

When one portion of the bowel invaginates into the immediately adjacent loop, the condition is called as an intussusception. Most of the time, it is proximal segment of bowel that invaginates into the distal segment. It is one of the commonest cause in paediatric age group. It can also occur in adults. In adults, our 2/3rd rule may be applied. Two-thirds of cases of adult intussusceptions are from known causes. Of these two-thirds are due to neoplasms. Of these neoplasms, two-thirds will be of malignant pathology.[24] An intussusception constitutes following parts:

- Intussusceptum
- Intussuscepiens
- Apex

- Neck

The types of intussusception are as follows:

Ileocolic (77%)

Ileo-ileocolic (12%)

Ileo-ileal (5%)

Colo-colic (2%)

Multiple (1%)

Retrograde (0.2%)

Others (2.8%)

In the paediatric age group, intussusception can occur in any age but more commonly in the period between 3 months to 9 months when the weaning from the breast milk is started. The incidence is higher in the first-born child. It is more common in prevalence in summer.

**Etiology**

Most of the cases of intussusception occur in the paediatric age group. Majority of the patients are classified as idiopathic and there may be association with acute gastroenteritis or URI , when the baby gets weaned from breast milk, it is believed to be due to hyperplasia of Peyer's patches in the terminal ileum, polyps, Meckel's diverticulum, duplication. H.S. Purpura or appendix as the common lead points. Most patients with adult intussusception are benign and enteric origin and most sensitive diagnostic modality is abdominal CT scanning.25 Large bowel intussusception is more common in adults. Half of the cases are secondary because of neoplasia either benign or malignant. Among benign conditions, fibroma, leiomyoma, polyps and submucous lipoma, Peutz Jeghers syndrome are the commonest one.

**Clinical features:** The typical presentation is with intermittent abdominal pain. Usually it is of sudden onset. During the attacks of pain the child cries with pallor over the face and becomes quiet after few minutes. Vomiting and blood stained

stools may be present. On examination, during pain free period a sausage shaped lump may be palpable with concavity towards umbilicus, which becomes hard on palpation. Right iliac fossa on palpation may be peculiarly empty (sign-de-dance). Patient may pass a few normal stools before 'red currant jelly' stool is passed.

## Radiography

Plain X-ray shows multiple air fluid levels. Barium enema in ileo-colic type shows the typical 'pincer shaped' or 'coiled spring' deformity or 'pinch fork' sign.

## Treatment

Intravenous fluid administration should be begun, decompression of small bowel through nasogastric suction is achieved. The reduction of the intussusception can be done by any of the following ways which may be non-operative reductions: Hydrostatic or pneumatic.

## Operative management

Laparotomy is done by making right lower paramedian incision. Intussusception mass is identified and a trial of manual reduction is attempted. The reduction is effected by squeezing the apex of the intususception retrogradely, but at no time the segments are pulled. Open method of inserting the little fingers into the neck and separating the adhesions between the intussusception in difficult cases can be tried (Copes method).

## Indications for resection

When there is presence of a polyp, tumour or Meckel's diverticulum or if the intussusception could not be reduced by manipulative procedures or if a frankly

gangrenous bowel is there, intestine should be resected without attempting for reduction.

The whole intussusception is usually resected with primary ileocolostomy. The ileum is resected proximal to the intussusception and the proximal end is anastomosed end to side with the transverse colon which is distal to the intussusception.

## VOLVULUS

Volvulus refers to the axial rotation of a portion of alimentary tract on its mesentery. It may be either primary or secondary. Primary volvulus can occur due to malrotation of the gut, abnormal attachment of the mesentry or congenital bands. E.g. Caecal volvulus, volvulus neonatorum and sigmoid volvulus.

A secondary volvulus is due to the actual rotation of a segment of bowel around an acquired adhesion or a stoma. When it is completed, it forms a close loop of obstruction with ischaemia of the involved segment.

## PRIMARY VOLVULUS

### Volvulus neonatorum

This condition is predisposed by arrested rotation of the gut causing the narrow mesentery of small bowel and caecum to get obstructed. Symptoms and

signs are abdominal pain, bilious vomiting. On laparotomy, the entire midgut has to be delivered out. Volvulus usually occurs in the clockwise direction, which has to be untwisted and if any secondary obstructing causes are present such as congenital bands (transduodenal band of ladd) have to be released and Ladd procedure can be performed. In this procedure, duodenum and the upper jejunum are repositioned on the right side of the abdomen.

**Caecal volvulus**

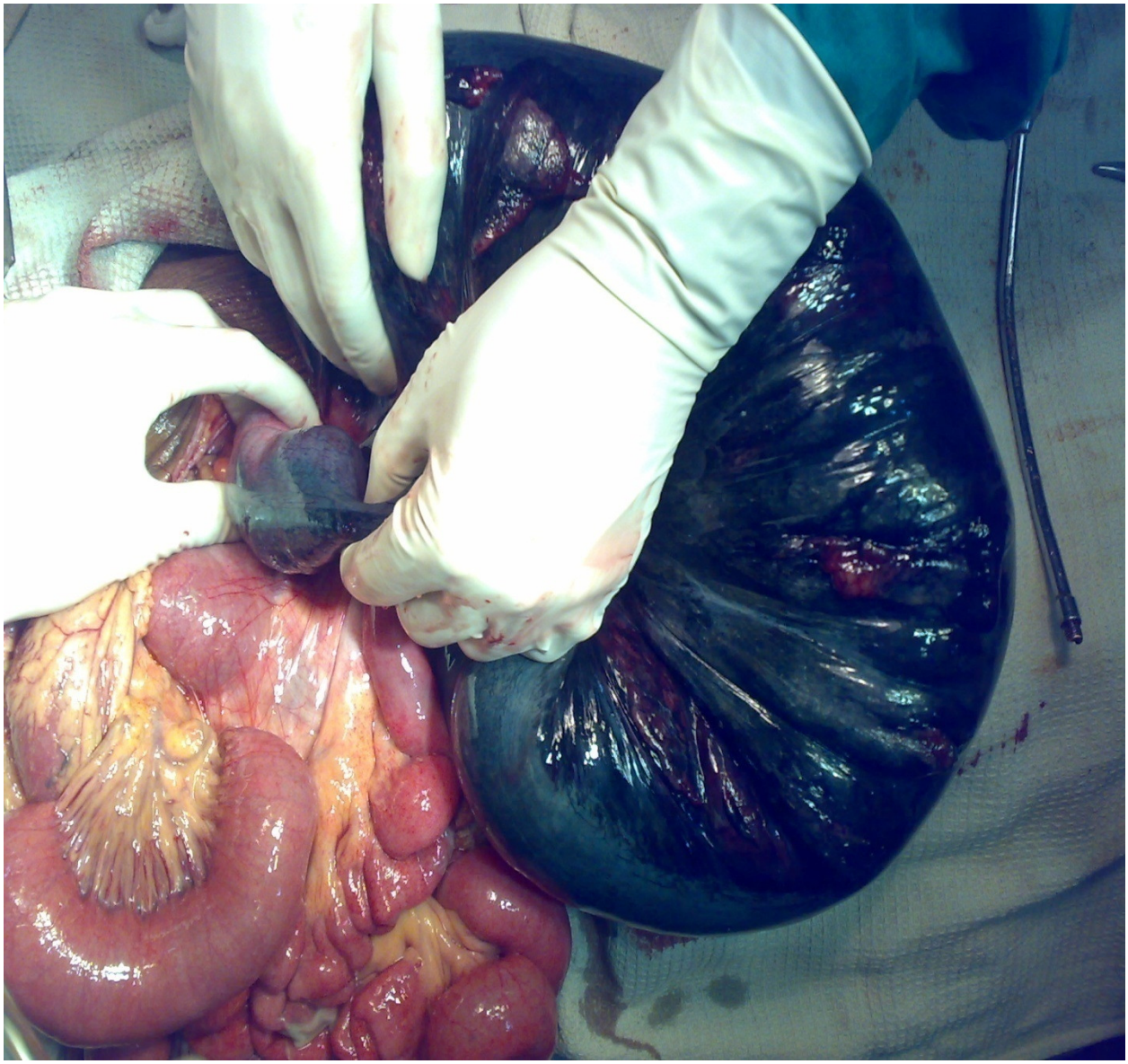
It may occur when the right half of colon is lax and mobile. It is more common in females with the classical features of obstruction like abdominal pain, nausea, vomiting, and constipation. At first, it may be a partial rotation and may spontaneously get released by passage of flatus and faeces. Barium enema will show the "bird beak" appearance and also absence of barium in the caecum. At laparotomy, volvulus may be treated by caecal needle decompression which can be followed by fixation of caecum to right iliac fossa or when the caecum is gangrenous or perforated, a right hemicolectomy is done.

**Sigmoid volvulus[19]**

Sigmoid volvulus is a serious entity in which, a redundant sigmoid loop rotates around its narrow and elongated mesentry, producing ischaemia and subsequential necrosis of the sigmoid colon, followed by rapid distension of the

closed loop of bowel.26 Volvulus of the pelvic colon is more common in the Eastern Europe, Russia and africa. It is rare in the USA and Western Europe. In India, it is commoner in the northern states like Punjab, UP, MP, Bihar and Maharashtra. This is the rotation of sigmoid loop of the colon over its mesentery. It commonly occurs in adults and in elderly males and is one of the commonest causes of large intestinal obstruction.

Aetiology of the condition includes a high residue diet with physical inactivity, chronic constipation, psychiatric problems, prolonged usage of sedatives, addiction to laxatives and acute antipsychotic drugs. Males are far more affected who may be middle aged or elderly. It is more prevalent in under-developed and malnourished people. Pathology may be due to a long pelvic mesocolon, adhesions over the sigmoid colon, a overloaded pelvic colon. The twist has odd turns, ½, 1½, 2½, etc. After the loop gets rotated 1½ turns, the veins which are involved in the torsion are compressed and the loop becomes massively congested and if the rotation is greater than 1½ times, loop becomes gangrenous. It presents with signs and symptoms suggestive of acute large bowel obstruction, which may be intermittent initially and followed by the passage of large quantity of faeces and flatus. Plain radiography of the abdomen reveals an inverted "U" shaped shadow of the distended intestinal loop (or coffee bean appearance) that runs from the right to left with two fluid levels, one in each loop.



**GANGRENOUS SIGMOID VOLVULUS**

## Management

Consists of either conservative or surgical measures. Surgery is mandatory, if gangrene is expected, resection and anastomosis has to be carried out.

## Conservative treatment

Sigmoidoscopy should be done, if the obstruction has reached a soft rectal tube is attempted to be put into the twisted gut. This will immediately cause deflation of the gut and surgery can be delayed until the patient becomes fit for surgery.

a. Oral medication.

b. Enema

## Surgical treatment

In patients with sigmoid volvulus, who do not have features of peritonitis on presentation, recurrence preventing surgery can be performed with minimal mortality rates.[28] After laparotomy through a midline or left paramedian incision, the loop may be untwisted and deflation of the distended loop can be done through a single stab incision under cover of a purse string suture. The gangrenous bowel must be resected and following procedure can be done.

a. Primary anastamosis after resection of the gangrenous segment with proximal covering colostomy.
b. A Paul Mickulicz type of double barrel colostomy.
c. Hartmann's procedure: Proximal end colostomy with closure of the end of the distal colon or rectum is done in two layers and is dropped into the abdominal cavity. Late anastamosis can be done after one month.

**Resective procedure**

a. Resection combined with Paul Mickulicz procedure or Hartmann's procedure followed by delayed anastamosis.
b. Resection and primary anastamosis: The redundant loop of the sigmoid colon can be resected and primary anastamosis can be done.

The mortality of patients with sigmoid volvulus who are treated surgically is closely related to the disease stage, appropriate surgical timing, the patient functional status and his collaboration with the clinicians is essential in order to define a correct diagnosis and the treatment.[28]

**Non-resective procedures**

a. Mesocolopexy: Plication of sigmoid colon can be done by various methods, which make the long mesocolon into a more broad based and shorter length, which avoids the torsion of the sigmoid colon on the mesentery Hall and Cragg's type, Prasad and Tiwari type have been advocated.
b. Sigmoidopexy: Involves fixation of the sigmoid loop to the posterior wall of the abdomen or to the parietal peritoneum or to transverse colon. The principle behind fixing the loop of sigmoid colon to various places is to curtail its free movements, which will avoid its twisting.
c. Extraperitonealization of the sigmoid colon: The sigmoid loop is placed in the space between the lower abdominal peritoneum and the abdominal musculature which may prevent or avoid rotation movements of the alimentary tract.

**SIMPLE VERSUS STRANGULATING OBSTRUCTION**

Obstructed external hernia is the second most common cause of intestinal obstruction. Most patients with small intestinal obstruction are classified as having simple obstruction that involve mechanical blockage of the flow of luminal contents without compromised viability of the bowel wall. In contrast, strangulating obstruction is the obstruction with compromised blood flow, it occurs

in nearly 25% patients with small intestinal obstruction. Various serum assays, including lactate dehydrogenase, amylase, alkaline phosphatase, and the ammonia levels, have been assessed without any real benefit. Initial reports have described some success in discriminating strangulation by measuring serum, creatine phosphokinase isoenzyme (particularly the BB isoenzyme), or intestinal fatty acid–binding protein; however, these are only investigational and could not be widely applied to patients with obstruction. Finally, non-invasive determinations of mesenteric ischemia are described using a super-conducting quantum interference device (SQUID) magnetometer to non-invasively detect mesenteric ischemia. Bowel ischemia is associated with changes in basic electrical rhythm of the small bowel. It is important to remember that bowel ischemia and strangulation cannot be reliably diagnosed or excluded pre-operatively in all cases by any known clinical parameters, combination of parameters, or current laboratory and radiographic examinations.

**Treatment**

Fluid Resuscitation and Antibiotics..

**Tube Decompression**

Apart from IV fluid therapy, another important adjunct to the supportive therapy of the patients with bowel obstruction is nasogastric suctioning.

Nasogastric suctioning with a Levin's tube causing emptying of the stomach, reducing the hazard of pulmonary aspiration of the vomitus and further minimizing bowel distention from preoperatively swallowed air. The use of long intestinal tubes (e.g., Cantor or Baker tubes) can be used. Patients with a partial intestinal obstruction can be treated conservatively with resuscitation and tube decompression alone. Enteroclysis can assist in determining the degree of obstruction, with higher-grade partial obstructions needing earlier surgical intervention. Although an initial trial of non-operative management of most patients with partial small bowel obstruction is required, it should be emphasized that the clinical deterioration of the patient or increasing small bowel distention on abdominal x-rays during tube decompression warrants prompt operative intervention.

**Operative Management:**

The patient with complete small bowel obstruction requires surgical intervention. A non-operative approach to the selected patients with complete small bowel obstruction has been proposed by some surgeons, who argue that prolonged intubation is safer in these patients provided that no fever, tachycardia, tenderness, or leucocytosis is noted. Nevertheless, one must realize that non-operative management of these patients is undertaken with a calculated risk of overlooking an underlying strangulation obstruction and delaying the management

of intestinal strangulation until after the injury has become irreversible. The Patients with intestinal obstruction secondary due to an adhesive band can be treated with lysis of the adhesions. Great care must be used in the gentle handling of the intestine to reduce the serosal trauma and avoid unnecessary dissection and inadvertent enterotomies. The hernia may be a spontaneous groin, epigastric or para-umbilical hernia or it may be an incisional or para-stomal hernia, the surgical approach and the initial dissection is similar to that in elective hernia repair.[32] Incarcerated hernias can be treated by manual reduction of the herniated segment of intestine and closure of the defect. The treatment of the patients with an obstruction and a history of malignancies can be managed by making a simple by-pass of the obstructing lesion, by whatever means, may offer the best alternative rather than a long and a complicated surgery that may entail intestinal resection. An obstruction which secondary to Crohn's disease will often subside with conservative treatment, if the obstruction is acute. If a chronic fibrotic stricture is the cause of the obstruction, then a intestinal resection or stricturoplasty may be needed. At the time of operative exploration, it can be sometimes difficult to assess intestinal viability after the release of a strangulation. If viability of the intestine is questionable, the bowel segment should be totally released and placed in a warm, saline-soaked sponge for 15 to 20 minutes and then re-examined. If the normal color has returned and peristalsis is evident, it is safe to retain the intestine.

In difficult borderline patients, fluorescein may supplement the clinical judgment. Alternative approach to the assessment of intestinal viability is the so-called second look laparotomy within 18 to 24 hours.

## TUBERCULOSIS OF INTESTINE

Intestinal obstruction is one of the most common complication in the small bowel, Affecting around 60% of the patients with tuberculous enteritis. Common sites are ileum, proximal colon and peritoneum. Approximately, 75% of patients with tuberculous enteritis have involvement of the distal small intestine and ileocaecal region.[33] Intestinal obstruction is the most common complication in the small intestine, affecting 60% of the patients with tuberculous enteritis.

There are two principal types:

1. Hyperplastic tuberculosis

2. Ulcerative tuberculosis:

## GALL STONE ILEUS

Gall stone ileus accounts for 1-2% of the cases with intestinal obstruction. It usually occurs in older age group. To cause obstruction, gall stone must be larger than 2.5 cm and it enters the intestinal tract by a process of ulceration. The stone passes through duodenum, jejunum and the colon. Obstruction is a cause at distal

ileum or at other areas of narrowing. The diagnosis can be done by the presence of air in the biliary tract along with the signs of acute intestinal obstruction in scout film. Management is removal of the stone via enterotomy or resection, if the stone is severely impacted.

**NEOPLASMS**

Extrinsic tumour involvement from secondary spread is the more likely cause of obstruction. Intestinal obstruction from metastatic disease arises when a loop of intestine gets trapped within the malignant masses. Carcinoma of the ovary, colon, stomach and the pancreas are the most common causes of this type of obstruction. Primary tumours of small intestine can cause intestinal obstruction either by obstructing the intestinal lumen or by acting as a nidus for intussusception. Although benign tumours can predispose conditions for intussusception, malignant tumours like adenocarcinoma, lymphomas and carcinoids can rarely give rise to obstruction, wide resection and end-to-end anastomosis is the treatment of choice. Foreign bodies and bezoars Luminal obstruction by the ingestion of foreign body is commonly noted in children and psychotic patients. Bezoars may migrate into the small intestine causing obstruction. Small intestinal obstruction can occur from bezoar arising from intestinal diverticulum. Bezoar or foreign body usually get impacted at the site where the bowel is narrowed by any previous surgery. Treatment is removal of the foreign body or bezoar by enterotomy.

# LARGE BOWEL OBSTRUCTION

Colorectal carcinoma is the single most common cause of large bowel obstruction in the United States, whereas colonic volvulus is the most common cause in Russia, Eastern Europe, and Africa. Intraluminal causes of colorectal obstruction include faecal impaction, inspissated barium, and foreign bodies. Intramural causes, include inflammation (diverticulitis, Crohn's disease, lymphogranuloma venereum, tuberculosis, and schistosomiasis), Hirschsprung's disease (aganglionosis), ischemia, radiation, intussusception, and any anastamotic stricture. Extraluminal causes include adhesions (the most common cause of small bowel obstruction, but rarely can cause colonic obstruction), hernias, tumors in the adjacent organs, abscesses, and volvulus.

## Etiology

Carcinoma of the colon is the most frequent cause of large-bowel obstruction in the developed countries . The left colon is the most likely place of obstruction and the extraperitoneal rectum being the least common. Signs of partial bowel obstruction progress to those of complete obstruction when the narrowed colonic lumen gets occluded by a fecal bolus. Since the right colon has semisolid contents and a relatively wider lumen, obstruction occurs late in this segment and may be acute in

its presentation, especially if the ileo-caecal valve is competent. The operative risk is elevated considerably when a perforation is present.

## SIGNS AND SYMPTOMS

The signs and symptoms of the large bowel obstruction mainly depend on the cause and location of the intestinal obstruction. Carcinoma arising in the rectum or the left colon are more likely to obstruct than those arising in the more capacious proximal large intestine. Regardless of the cause of the block, the clinical manifestations of large intestinal obstruction include failure to pass stools and flatus associated with an increasing abdominal distention and crampy abdominal pain. Colonic obstruction is often associated with potentially serious complications such as perforation, only 4% of the tumours of the colon present with the perforation of the intestine, and the timing and selection of appropriate operative procedures are also important. Symptoms tend to develop slowly and progressively or may develop fulminantly. Among adults, the elderly people are usually affected. The sigmoid colon is the usual site: this portion of the bowel is thick walled, not particularly distensible, and comparatively narrow.

## MANAGEMENT

All patients with complete acute large intestinal obstruction, require prompt surgical intervention and should not undergo a trial of non-operative management.

Naso-gastric decompression is also important in patients with large intestinal obstruction to reduce the amount of air and gastric contents delivered into the bowel. Nasogastric decompression will help to relieve intraluminal pressure, prevent further dilatation of the proximal intestine, and possibly decrease the risk of perforation. Antibiotics targeted at both the skin and the colonic flora should be administered. Exploration in patients with large intestinal obstructions is best performed through a lower midline incision. Patients with large intestinal obstructions should be placed in the lithotomy or modified lithotomy positions if access to the anus is anticipated. Obstructing lesions of the caecum and the ascending colon should be resected through a right hemicolectomy, usually with a primary anastamosis. Lesions in the transverse colon could be managed with an extended right hemicolectomy and again, with a primary anastamosis. Proximal diversion with an end ileostomy is usually not necessary in all patients; however, proximal diversion can be considered when there is any concern about intestinal viability, if the patient is unstable, or in the presence of substantial peritoneal contamination or peritonitis.

The treatment of obstructing lesions in the descending and the sigmoid colon is a more classical approach with Hartmann's procedure of segmental resection of the affected colon, an end colostomy, and a blind distal pouch or a mucous fistula. An end colostomy at the time of surgery is safe and may reduce the incidence of

perioperative complications when compared to an on-the-table bowel preparation with primary anastamosis. Another option to be considered in the early management of the patient with an obstructing lesion in the large intestine is the use of a self-expanding intraluminal metal stent (SEMS) to allow for immediate colonic decompression and the ability to perform elective mechanical bowel preparation. The usage of SEMS is becoming widely available and it can be an useful tool for the surgeon managing a large intestinal obstruction. In experienced hands, a SEMS may be placed successfully in about 90% of the patients with low complication rates. A SEMS can avoid the need for an urgent or emergent operation by intra-luminally decompressing the distended proximal colon and allowing for distal passage of stool. A SEMS is also useful when palliating patients who could not tolerate surgical diversion or those with an unresectable disease and a limited survival. With a locally advanced obstructing rectal carcinoma, after placement of SEMS, the patient can be given neo-adjuvant therapy followed by surgical resection, again increasing the probability for a successful one-stage operation. The cause of the obstruction needs to be treated individually. Thus a hernia is repaired when the obstructed colon is reduced, whereas an intussusception is relieved, with resection of the involved large bowel if necessary. Because it is so common, fecal impaction could be ruled out in the institutionalized or debilitated elderly patients. The caecum must be visualized to

assess its viability. If an emergency anastamosis is to be performed, the criteria for a better outcome must be met.

## ADYNAMIC OBSTRUCTION

Adynamic obstruction, in which peristalsis may be absent as in paralytic ileus or it may be present in a non propulsive form as in mesenteric vascular obstruction or pseudo-obstruction.

Causes of Ileus

- Idiopathic
- After laparotomy
- Metabolic and electrolyte abnormalities (e.g., hypokalemia, hyponatremia, hypomagnesemia, uremia, diabetic coma)
- Drugs (e.g., opiates, psychotropic agents, anti-cholinergic agents)
- Intra-abdominal inflammation
- Retro-peritoneal hemorrhage or inflammation
- Bowel ischemia
- Burns

• Strokes

Patients often present in a pattern similar to those with a mechanical small intestinal obstruction. Abdominal distention, usually without colicky abdominal pain, is the typical and the most notable finding. Nausea and vomiting can occur but may also be absent. Patients with an ileus can continue to pass flatus and diarrhea, and this may help to distinguish these cases from those with a mechanical small intestinal obstruction.

Radiologic studies may help in distinguishing ileus from small intestinal obstruction. Plain abdominal radiographs may show distended small bowel as well as large intestinal loops. The treatment of ileus is entirely supportive with nasogastric decompression and parenteral fluids. The most effective management to correct the underlying condition.

**Mesenteric Vascular Occlusion**

Arterial embolism is commoner than spontaneous thrombosis and the superior mesenteric vessels have been implicated more frequently. Possible sources of emboli include atrial fibrillation, a mural myocardial infarct, an atheromatous plaque or aneurysm, a vegetation of the mitral valve, pulmonary vein thrombosis.

The ischaemia of the bowel may be due to arterial or venous sources, the intestines and its mesentery. The intestines and its mesentery may become swollen and

oedematous, demarcation between healthy and the infarcted bowel being gradual. Blood stained fluid is exuded into the peritoneal cavity and the lumen of the infarcted intestine, which gets filled with blood. Acute mesenteric ischaemia is a highly morbid event with reported mortality rate exceeding 60%. When the main branch of the superior mesenteric artery is occluded, the whole of the small bowel, caecum and a part of the ascending colon may become infarcted.

Mesenteric vascular occlusion must be suspected, when a patient beyond the middle age, giving a history of cardiac illness, it is suddenly associated with acute abdominal pain that is not colicky in character. He may collapse and the patient may pass blood stained stools.

Clinical features

(i) Pain which may be central abdominal in nature.

(ii) Delayed Gastrointestinal emptying with persistent vomiting.

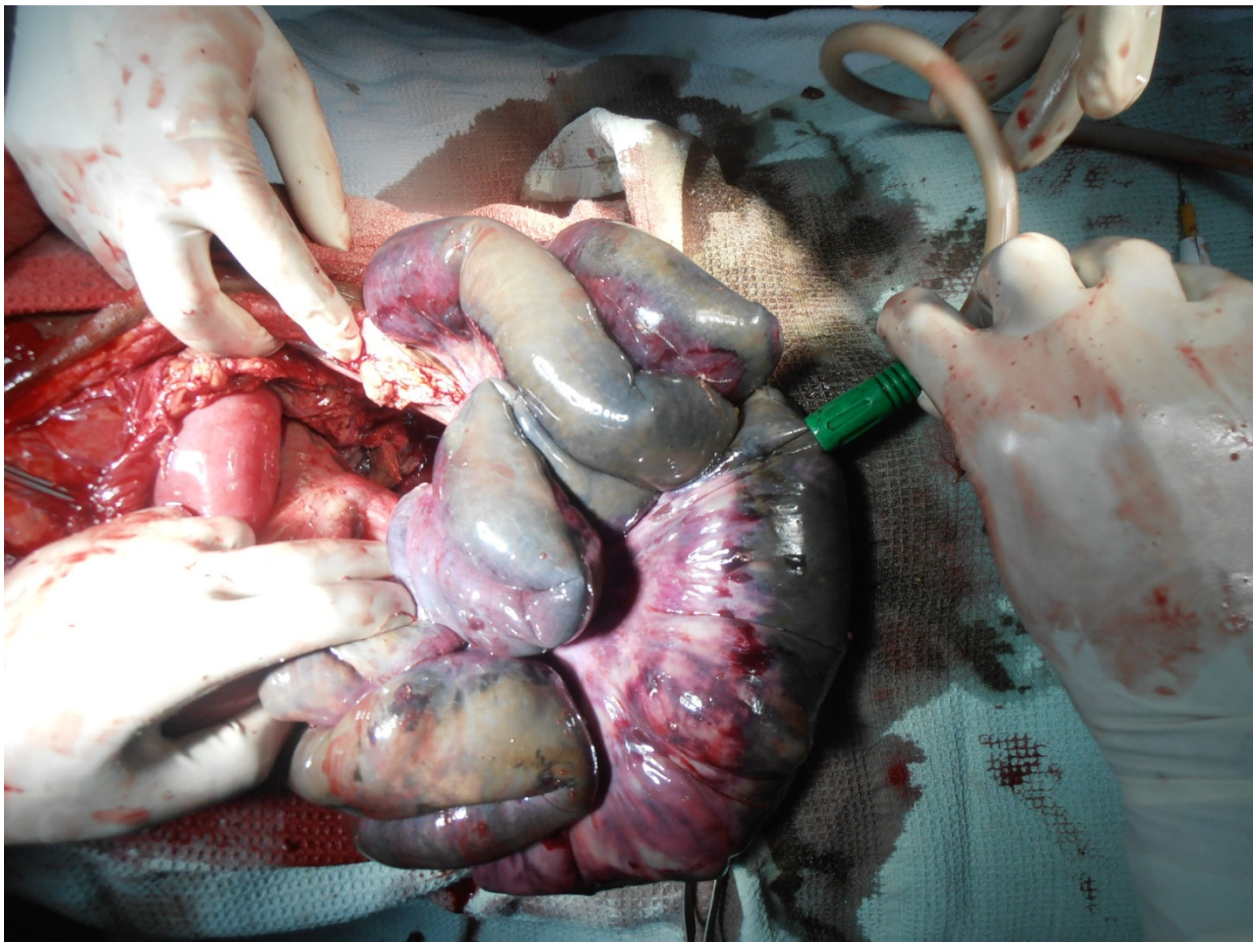
Investigations

Mesenteric angiography is the definitive diagnostic study. Duplex ultra- sonogram may be of some benefit in visualizing the blood-flow in the SMA. Ogata et al. reported that a kinetic dilated bowel loop observed on the real time ultrasonography has a high sensitivity 90% and the specificity 93% for the

recognition of strangulation. The positive predictive value is 73%.1 CT abdomen and pelvis reveals focal or segmental intestinal wall thickening.39

Treatment

Regardless of the aetiology, the prognosis of the patient with mesenteric ischaemia is dependent upon the rapid diagnosis and initiation of management. Conservative management may be sufficient in selected patients;  more often laparotomy may be required and can be life saving.40 Superior mesenteric artery embolectomy should be attempted and thrombo- endarterectomy or a bypass procedure from aorta or iliac arteries or the splenic artery to the more distal artery in mesenteric vascular tree. In late cases, the affected gangrenous intestine should be resected.



**SMALL BOWEL GANGRENE**

# METHODOLOGY

The materials for this clinical study on intestinal obstruction were collected from cases admitted to various surgical wards in Thanjavur medical college Hospital attached to Thanjavur Medical College , Thanjavur, during the period from 1st March 2012 to 31st December 2013, fifty cases of intestinal obstruction have been studied. Patients belonged to the age group ranging from 12 years to 85 years, paediatric age group being excluded from this study. The criteria for selection of cases was based on the clinical history, physical examination findings, radiological and haematological investigations.

Patients who had subacute Intestinal obstruction , who were treated conservatively were excluded from the study, and only those patients of acute intestinal obstruction which were managed surgically have been studied to establish the pathology of intestinal obstruction with an aim to identify the mode of presentation, physical findings, radiological and haematological findings, operative findings and outcome of acute bowel obstruction. After admission of the patient, clinical data were recorded according to the Proforma. The diagnosis was mainly based on clinical examination and often supported by haematological and radiological examinations.

## Methods

Study has been divided into

a. Clinical study

b. Investigations

c. Treatment

Study has been conducted under the following headings:

a. History taking

b. Physical examination

c. Laboratory examination

d. Radiological examination – Plain X-ray abdomen – erect view.

e. Ultrasound examination in selected cases

f. Surgical treatment and results

g. Follow-up

a. History taking

A complete detailed history was obtained from the patient and the complaints were entered in the proforma in a chronological order. Each complaint in the history of presenting illness has been documented in detailed enquiry.

b. physical examination

(i) General physical examination – evidence of dehydration and its severity were looked into and vital parameters were recorded.

(ii) Local examination – Abdominal examination was done under the standard headings inspection, palpation, percussion and auscultation. Per rectal examination was done and findings were noted.

(iii) Systemic examination – All other systems were examined carefully to rule out any associated anomalies and to assess the fitness for surgery.

c. Laboratory examination

(i) Haemoglobin

(ii) TC & DC

(iii) Bleeding and clotting times

(iv) Blood grouping and Rh typing

(v) Urine for albumin estimation and microscopy

d. Radiological examination

Erect abdomen X-ray is done in all cases, barium enema and ultrasound examination is done in selected cases.

**SURGICAL MANAGEMENT**

Immediately after the admission along with above procedure, resuscitation with IV fluids especially ringer lactate and normal saline infusion is started till the hydration and urine output become normal. Nasogastric decompression with Ryles tube insertion is carried out and antibiotic prophylaxis is started. A close observation of all bedside parameters (like pulse rate, BP, RR, urine output, urine output, abdominal girth, bowel sounds and tenderness and guarding) was done. Emergency Blood transfusion was given in required cases. Patients who showed a reduction in the abdominal distension and improvement in the general condition especially in those with postoperative adhesions, conservative treatment was confined (by extending the supportive treatment) for next 24 hours, those who showed improvement by moving bowels or reduction in pain/tenderness were considered for further conservative treatment and such individuals are excluded from this study. Patients with clear-cut signs and symptoms of acute obstruction had been managed by appropriate surgical procedure after initial resuscitation. Surgery adopted and the criteria for deciding the procedure were noted, e.g. release

of a band or an adhesion, reduction and caecopexy for intussusception, resection and anastamosis for gangrenous intestine and release and repair for strangulated obstruction. Histopathological examination of the specimen of resection/biopsy was undertaken whenever necessary.

The postoperative period had been monitored carefully and all the parameters were recorded hourly or fourth hourly basis depending on the patient's general condition and toxemia. Postoperatively Nasogastric tube aspiration, intravenous fluids and antibiotics were administered. Any complications were noted and treated accordingly.

Postoperative follow-up after the discharge of patients was done in majority of the patients till 6 months. Most of the patients did not turn up for follow up after one or two visits. The results are tabulated stressing on the following points like age, sex, symptoms, examination findings, investigations, abnormalities, possible causative factors, operative findings and operative procedure that is adopted and complications if any.

Statistical Methods: Chi-square and Fisher Exact test has been used to find the significance of proportion of Postoperative complications in association with etiology of acute Intestinal Obstruction.

1. Chi-Square Test

$$\chi^2 = \frac{\sum (Oi - Ei)^2}{Ei}$$

, Where Oi is observed frequency and Ei is Expected frequency

Statistical software

The Statistical software namely SPSS 11.0 and Systat 8.0 had been used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables, etc.

# RESULTS

The incidence of acute bowel obstruction in adult age group was studied from the cases admitted in Department of Surgery of Thanjavur Medical College Hospital attached to the thanjavur Medical College, Thanjavur during the period 1st March 2012 to 31st December 2013. The data on the symptoms and the signs and laboratory investigations has been adopted in 50 cases during this study period. During the period of 20 months, the total number of admissions in surgery were 12,233 patients. Of which 228 cases with acute intestinal obstruction were treated during this period which comprise 1.9% of the total admissions. Among these surgically managed cases, 50 cases were randomly selected for the present study.

Table 1: Age incidence

| **Age (years)** | **Male** | **Female** | **Total** |
|---|---|---|---|
| 11 to 20 | 5 | 1 | 6 |
| 21 to 30 | 5 | 3 | 8 |
| 31 to 40 | 7 | 3 | 10 |
| 41 to 50 | 3 | 1 | 4 |
| 51 to 60 | 8 | 2 | 10 |
| 61 to 70 | 5 | 3 | 8 |
| 71 to 80 | 2 | 1 | 3 |
| 81 to 90 | 1 | 0 | 1 |
| Total | 36 | 14 | 50 |

Chart 1 AGE INCIDENCE

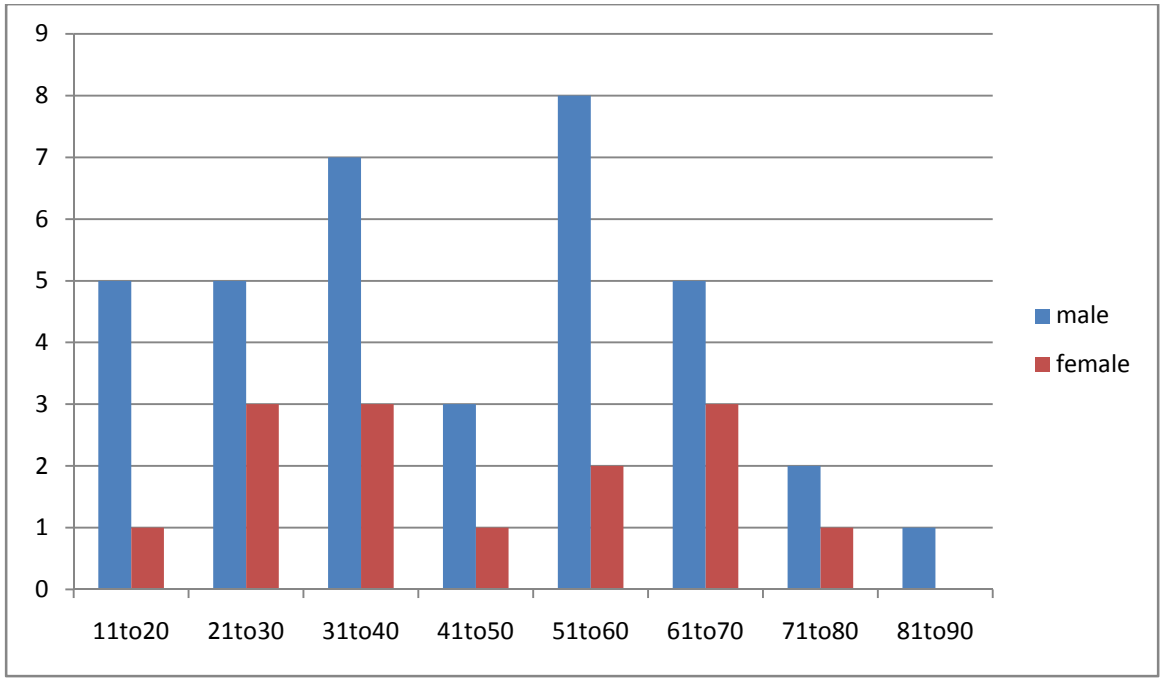
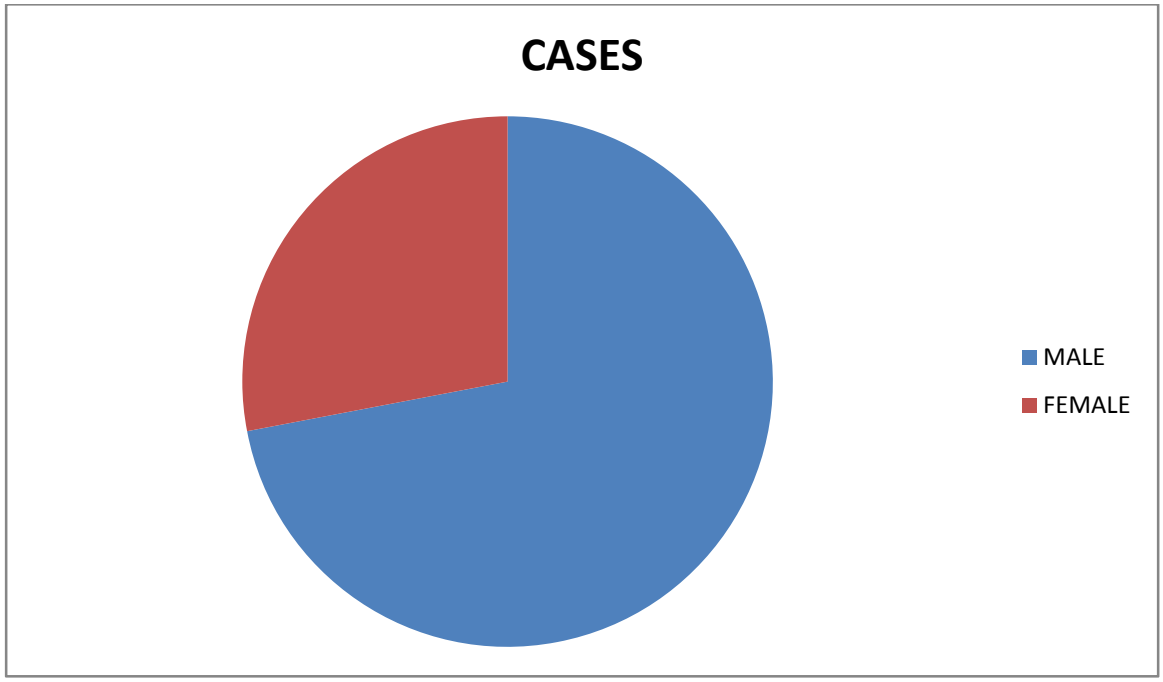



CHART 2 :Sex incidence



According to the above table and bar chart, the peak incidence in the present study group is 31-40 and 51-60 with each consisting of 10 cases out of 50 cases.

**2: Sex incidence**

| Sex | Number of cases | Percentage |
|---|---|---|
| Male | 36 | 72 |
| Female | 14 | 28 |

Male patients were more commonly affected when compared with females in the ratio of 4:1 in the above table.

Table 3: Socio-economic status

| Socio-economic status | Number of cases | Percentage |
|---|---|---|
| Poor | 38 | 76 |
| Middle | 12 | 24 |
| Upper | 0 | 0 |
| Total | 50 | 100 |

CHART 3: Socio-economic status

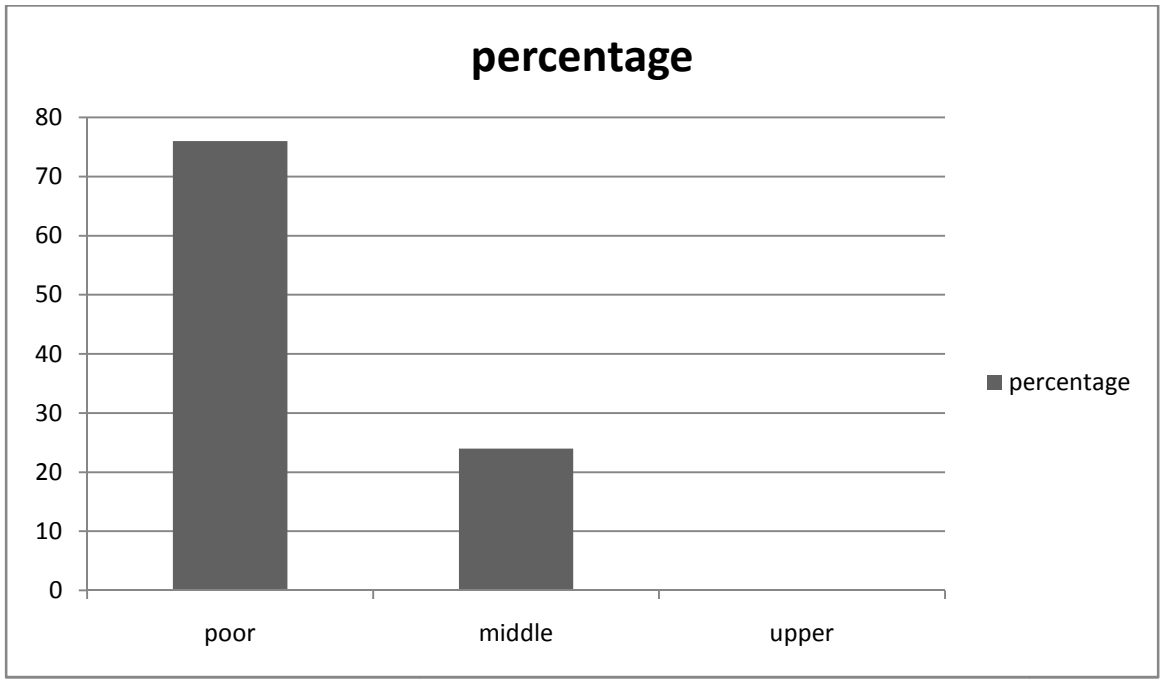



CHART 4 : Diet

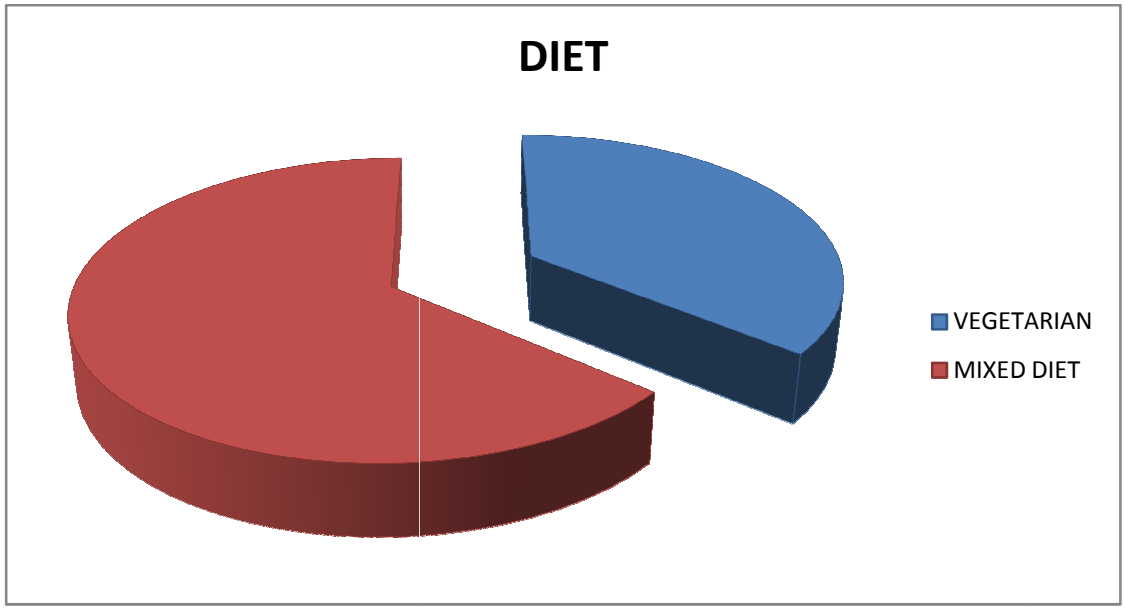


Table 4: Diet

| Diet | Number of cases | Percentage |
|---|---|---|
| Vegetarian | 18 | 36 |
| Non-vegetarian | 32 | 64 |
| Total | 50 | 100 |

In the present study consisting of 50 cases, 32 patients were taking non-vegetarian diet which contains more of fatty diets. The remaining 18 patients were vegetarian which often contained high fibre content.

Table -5 : Symptoms and signs

| Symptoms | Signs |
|---|---|
| Abdominal pain<br>Vomiting<br>Abd. Distension<br>Constipation | Tachycardia<br>Previous surgical scar<br>Tenderness<br>Rigidity<br>Mass<br>Visible peristalsis |

In The present study, the most common symptoms were pain abdomen (88%) andvomiting (78%), and the most common signs were tachycardia (80%) and visibleintestinal peristalsis (60%).

INCIDENCE OF DIFFERENT AETIOLOGY

The incidence of different etiologies of intestinal obstruction in the present series are as follows.

Table 6: Causes of intestinal obstruction in adults

| **Clinical condition** | **Number of cases** |
|---|---|
| Postoperative adhesions | 20 |
| Obstructed hernia | 15 |
| Volvulus | 2 |
| TB abdomen | 2 |
| Malignancy | 7 |
| Intussusception | 3 |
| Mesenteric ischaemia | 1 |
| Total | 50 |

CHART 6 : CAUSES

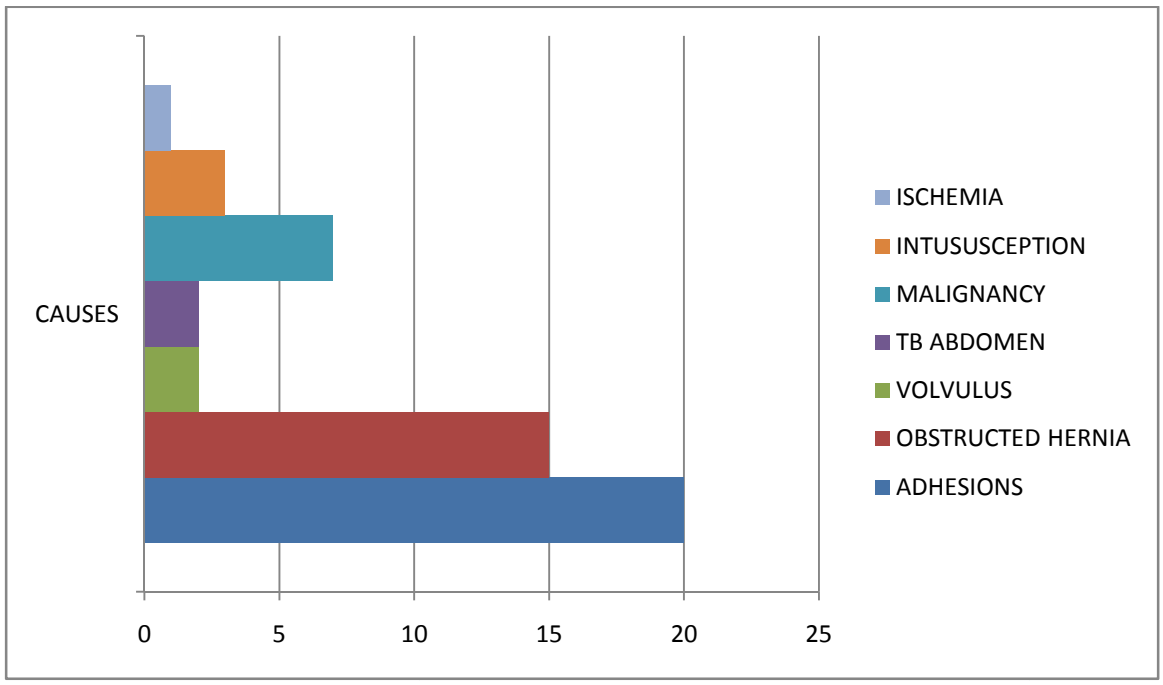
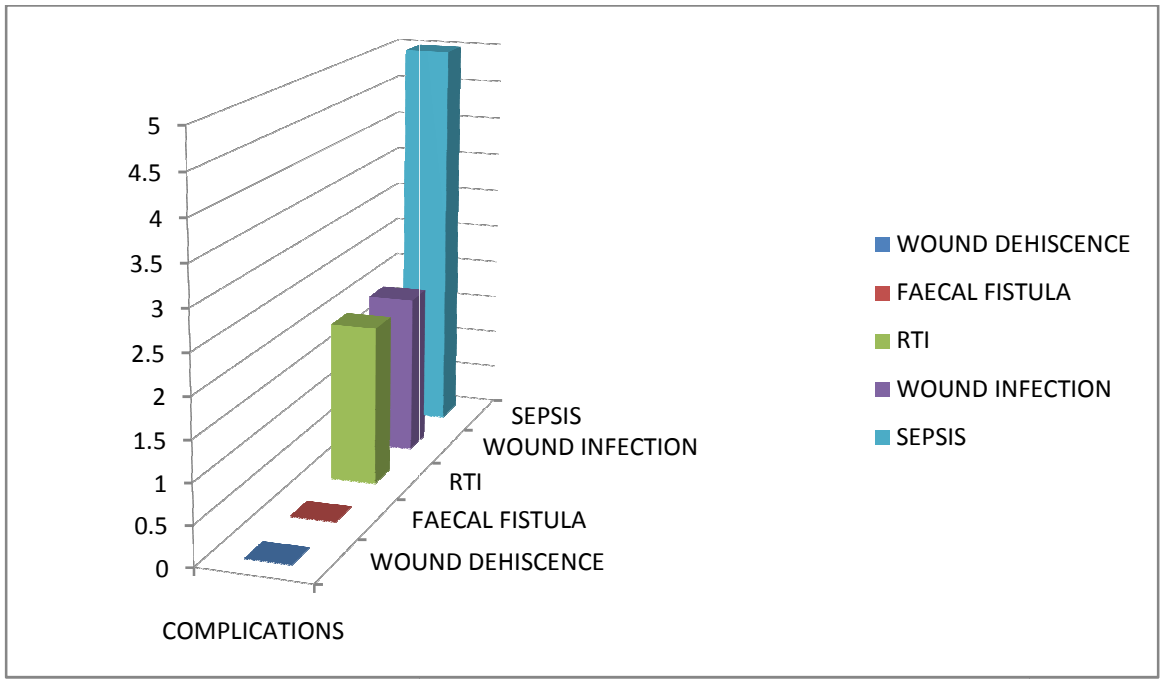



CHART 7: Postoperative complications



**Clinical conditions**

The most common cause of intestinal obstruction in this study was postoperative adhesions. The next common was obstructed hernia. Other conditions include volvulus, intussusception, tuberculosis, malignancy, mesenteric ischaemia, in the order of descending frequency.

**Management**

In our study of 50 cases as accordingly with the aetiology the management and the surgical procedure was done as shown in the table and pie diagram. Release of adhesions was done in 40% of cases, resection anastamosis in 22% of cases and release of adhesion with herniorraphy done in 18% of the cases.

**Post- operative complications:**

In the present study group there were 5 cases of septicemia, 2 cases of respiratory tract infection and 2 cases of wound infection.

Table 7: Postoperative complications

| Postoperative complications | |
|---|---|
| WI | 2 |
| RTI | 2 |
| Wound dehiscence | - |
| Faecal fistula | - |
| Septicaemia | 5 |

## Mortality

In the present study of 50 cases, about 7 patients died with the percentage of 14%. The majority of deaths due to complications like septicemia, peritonitis, respiratory infection. In the present study 7 persons died during postoperative period. The analysis of cause of death is shown below.

Table : 8 Mortality

| Mortality | No. of cases | percentage |
|---|---|---|
| Cured | 43 | 86 |
| Dead | 7 | 14 |

Table 9 : Causeof Death

| Age and sex | Symptoms prior to admission | Operative findings | Operative procedure | Cause of death |
|---|---|---|---|---|
| 75/F (Case No. 8) | 3 days | Carcinoma sigmoid colon | Resection and anastamosis | Septicaemic shock |
| 72/M (Case No.11) | 8 days | Carcinoma rectum | Hartman's procedure | RTI |
| 65/M (Case No.21) | 5 days | Mesenteric ischaemic | Resection anastamosis | Septicaemic shock |
| 45/M (Case No.36) | 3 days | Carcinoma caecum | Resection and anastamosis | RTI |
| 38/F (Case No. 37) | 5 days | Carcinoma ovary with sigmoid colon infiltration | Transverseloop colostomy | Septicaemic shock |
| 63/M (Case No.39) | 3 days | Carcinoma Rectum | Hartman's procedure | Septicaemia |
| 55/M (Case No.43) | 4 days | Carcinoma colon | Resection and anastamosis | Septicaemia |

# DISCUSSION

Acute intestinal obstruction persists to be the most common surgical emergency. In our study a total number of 12,233 patients were admitted in the surgery department from March 2012 to December 2013. A total number of 228 patients presented with features of acute bowel obstruction. Among these 50 of operated patients were randomly selected for the present study.

Disease Incidence

In our clinical study, the incidence of acute intestinal obstruction is 1.9% of the total surgical cases. In Souvik Adhikari et al. series, the incidence was 9.87% of total surgical cases. In Bhargava Anderson's series incidence was 3% of total number of surgical cases. The most common cause was found to be the postoperative adhesions followed by obstructed/ strangulated inguinal hernia, carcinoma, intussusception, volvulus, tuberculosis and mesenteric ischaemia. Although in the developing countries like India, the commonest cause used to be obstructed /strangulated hernia, in our study, commonest cause was adhesions followed by obstructed/strangulated hernia as the second cause. The decrease in the incidence of obstructed hernia indicate the changing trend towards early surgery before hernia gets complicated. The data of the present series is comparable to Souvik Adhikari series, Cole series and Jahangir-Sarwar Khan

series. Souvik Adhikari et al. (2010)44 reported an incidence of 9.87%, Bhargava and Anderson series reported an incidence of 3%. In our hospital 1569 cases of total emergency surgeries were done in march 2012 to December 2013, of which 228 cases with intestinal obstruction who comprise of 14.3% incidence were present. Among these 50 cases were selected as a random study group.

Age Incidence

Acute Intestinal obstruction although occurs in all age groups, the age spectrum in our study is 15 years to 85 years. The study showed the maximum incidence is in the age group 31-40 of 20% and 51-60 years of 20% which is comparable to the previous study groups Souvik Adhikari et al., Cole GJ et al. group, which are nearly similar to our clinical study of acute intestinal obstruction. The mean age is our current study is 45 years where as Souvik Adhikari et al.44 shows mean age of 44 years, Jahangir Sarwar Khan45 series shows a mean age of 33 years These studies are almost comparable with our current clinical study

Table 11: Age incidence of intestinal obstruction in different studies

| Age group | Cole GJ46 | Souvik Adhikari | Harban Singh | Present study |
|---|---|---|---|---|
| 12-20 | 10% | 9% | 10% | 12% |

| | | | | |
|---|---|---|---|---|
| 21-30 | 10% | 11% | 16% | 16% |
| 31-40 | 18% | 15% | 18% | 20% |
| 41-50 | 16% | 24% | 15% | 8% |
| 51-60 | 15% | 13% | 10% | 20% |
| 61-70 | 16% | 20% | 20% | 16% |
| 71-80 | 9% | 8% | 5% | 6% |

**Sex Incidence**

In the Souvik Adhikari et al.44 study, male to female radio was 4:1. In the Osuigwe ANet al. study, male to female ratio was 2:1. In the present clinical study male to female ratio is 4:1.

**Etiology**

The cause of acute intestinal obstruction differs from different geographical locations. In the present clinical study, about 76% of the patients were of poor socio- economic class and the remaining 24% were of middle class which does not yield much statistical significance. But our hospital being a government set-up, which is serving mostly the poor socio-economic status, hence the percentage of poor socio-economic status is high. The diet pattern in this study showed 64% to

be non-vegetarians and 36% to be vegetarians which did not show any significance in relation to the disease. In the present clinical study of 50 cases of acute intestinal obstruction, 40% of the cases occurred due to post operative adhesions who has undergone previous surgeries.

Table 12: Comparison of etiology with other studies

| Cause | Souvik adhikari | Jahangir | Brooks Arshad | Playforth49 | Cole And M. | Present study |
|---|---|---|---|---|---|---|
| Adhesions | 16% | 34% | | 3% | 5% | 5% |
| Hernia | 36% | 5% | 4% | 9% | 18% | 6% |
| Volvulus | 6% | 1% | 24% | 12% | - | 40% |
| Tuberculosis | 14% | 3% | 2% | - | 54% | 30% |
| Malignancy | 17% | 6% | - | 23% | 23% | 4% |
| Intussusception | 2% | 2% | 10% | 25% | 3% | 4% |
| Mes.Ischaemia | 9% | 41% | 10% | 1% | - | 14% |
| Miscellaneous | 49% | 19% | 35% | - | 9% | 6% |

In the present study, postoperative adhesions are the most common cause of intestinal obstruction, which can be comparable with the other study groups -

Playforth et al. with 54% and Arshad Malik et al. with around 41%. Although the incidence of obstructed/ strangulated hernia is more in developing countries, in this study group, it is the second most common aetiology for the intestinal obstruction. It may be because of the awareness of public, the availability of good surgical facilities in the periphery for the hernia repair, the hernias are managed early.

Clinical features

The clinical feature of intestinal obstruction like abdominal pain , vomiting Abdominal distension and constipation were not present in all cases. Pain abdomen was present in 88% of the patients in the present study, where as vomiting was present in 78% of the patients. Abdominal distension was present in 66% and constipation was present in 54% of the cases. The comparison table showing percentage of clinical features by various other study groups are as follows.

Table 13: Comparison of clinical features with other studies

| **Study group** | **Pain abdomen** | **Vomiting** | **Distension** | **Constipation** |
|---|---|---|---|---|
| Present study | 88% | 78% | 66% | 64% |
| Souvik | 72% | 91% | 93% | 82% |

| | | | | |
|---|---|---|---|---|
| Adhikari | | | | |
| Jahangir-Sarwar Khan | 100% | 92 | 97 | 97 |

In the present study, the clinical features of abdominal pain was 88%, vomiting was 78%, which comparable with the other study groups (Souvik Adhikari et al. and Jahangir Sarwar Khan et al). Only about 66% of the patients in the present study group had Abdominal distension. It may be due to an early approach to the hospital by patients in the present study. The abdominal mass on palpation is present in 24% of the total study, more in Malignancy and ileocaecal tuberculosis. Visible peristalsis is present in only 60% of the intestinal obstruction patients. The rectal examination did not reveal any abnormality except in four patients of intussusception (8%) and 2 cases of malignancy (4%) where in red currant Jelly and rectal growth were the rectal examination findings respectively.

**Surgical Management**

The surgical management in the present study group includes release of adhesions for postoperative adhesions 40%, resection and anastamosis for many of the cases of obstructed/strangulated hernia where the viability of the intestine was doubtful

and also for ischaemic bowel 22%, release of the constricting agents and herniorrhaphy was done in 18% of the obstructed/strangulated hernia cases. Derotation of the volvulus and sigmoidopexy was done in around 4% of the cases. Resection and anastamosis and herniorraphy was done in 8% of the cases. Reduction of intussusception was done in one case. Two cases were managed with Hartman's procedure and one patient with a transverse loop colostomy.

Complications

In the present group out of the 50 cases, complications like septicemia occurred in 5 cases, respiratory tract infection in 2 cases, wound infection occurred in two cases. The complication of septicemia was more in the patients with malignancy and one case with mesenteric ischaemia wherein there was already sepsis at the time of admission. Bowel surgeries were done in unprepared bowel in such cases. In Two cases – one with obstructed inguinal hernia and one with carcinoma rectum, the patients already had prior co-morbid conditions of COPD, and they suffered from respiratory tract infection.

**Mortality**

Frequency of mortality in this study is 14% i.e. 7 cases out of 50 cases. Among these, 6 cases were because of malignancy and one due to mesenteric ischaemia. Mortality that occurred during various studies have been tabulated as follows.

Table 14: Morality rate in various studies

| Studies | Year | No. of cases Studied | Mortality |
|---|---|---|---|
| Present study | 2009 | 50 | 14% |
| Souvik Adhikari | 2005 | 367 | 7.35% |
| Safian Matsu Moto | 1975 | 171 | 19% |
| Jahangir-Sarwar Khan | 2001 | 100 | 7% |
| Ramachandran | 1982 | 417 | 12.7% |

The mortality rate in the present study is comparable to the Ramachandran CS et al. study but it is more when compared to Souvik Adhikari et al., Jahangir et al. studies. Out of 7 cases who died, 6 cases were due to malignancy. As the malignancy was more in the aged group and with the unprepared bowel surgeries done to the patient, it led to septicemic condition and resulted in death. Two patients were chronic smokers who suffered respiratory tract infection and died. Hence most of the deaths were due to malignancy which played significant part in the outcome of the disease. The mortality with intestinal obstruction is more in patients who develop strangulation and gangrene of the bowel, also who reached the hospital after 3 days.

# CONCLUSION

• Acute intestinal obstruction remains to be an important surgical emergency in the surgical field.

• Success in the management of acute intestinal obstruction depends largely upon the early Diagnosis, skillful management and treating the pathological effects of the obstruction as much as the cause itself.

• Erect abdomen X-ray is a valuable investigation in the diagnosis of acute intestinal obstruction.

• Post-operative adhesions are the common cause to produce intestinal obstruction. Clinical, radiological and operative findings when put together can diagnose the intestinal obstruction.

• Mortality is still significantly high in case of acute intestinal obstruction.

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## PROFORMA

| | | |
|---|---|---|
| Sl.No. | Age: | Sex: |
| Name: | Occupation: | Address: |
| DOA: | DOD: | Unit : |

Economic status:

1. Chief Complaints:

2. History of Present Illness:

- Pain
- Vomiting
- Distension
- Bowel habits
- H/o passing blood in stools
- H/o fever
- H/o Jaundice

3.Past History:

- H/o tuberculosis
- H/o any surgeries
- H/o passing worms blood in stools
- H/o altered bowel habits

4. Personal History:

- Micturation
- Bowel habits
- Sleep
- Weight loss

- Appetite
- Smoker
- Alcoholic

5. Menstrual History (females):

6. Obstetric History (females):

7. Family History:

8.General Physical Examination:

Vital Signs

- Pallor:
- Cyanosis:
- Clubbing:
- Jaundice:
- Edema:
- LN:

Pulse:

Blood pressure:

Respiratory rare

Temperature

9.Systemic Examination:

- Shape:
- Distension:

Inspections

- Respiratory movements of each region:

- Peristaltic movements:
- Position of umbilicus:
- Scars:
- Any skin changes:
- Any mass:
- Hernial sites
- Renal angles:
- Supraclavicular fossa:

Palpation

- Cutaneous hyperaesthesia:
- Tenderness:
- Rebound tenderness:
- Muscular rigidity:
- Palpable coils of intestine:
- Is caecum palpable:
- Palpation of hernial orifice:
- Any mass palpable:
- Supraclavicular fossa:
- Testes:

Percussion

Auscultation

Per rectal

Per vaginal

Cardiovascular system

Respiratory system

Central nervous system:

10.Investigation:

Blood:

Hb%:

ESR:

TC:

Urea:

Sugar:

DC:

RBS:

BT:

Albumin:

CT:

Microscopy:

Blood grouping and Rh typing:

Urine:

ECG:

X-ray:

X-ray of the chest

X-ray of the abdomen (Erect):

11. Preoperative Diagnosis:

12.Treatment

Preoperative treatment

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Hourly | | | | | | | | |
| Pulse | | | | | | | | |
| Blood pressure | | | | | | | | |
| Temperature | | | | | | | | |
| Respiratory rate | | | | | | | | |
| IV fluids | | | | | | | | |
| RTA | | | | | | | | |
| UOP | | | | | | | | |
| Abdominal girth | | | | | | | | |
| Blood transfusion: | | | | | | | | |
| Drugs: | | | | | | | | |

13. Operative management

Anaesthesia:

Incision:

Gross appearance:

Small bowel:

Peritoneum

Colour:

Gangrene:

Perforation:

Colour:

Large bowel:

14. Pathology noted:

15. Surgical procedures:

16. Postoperative treatment:

| Days | | | | | | | |
|---|---|---|---|---|---|---|---|
| Pulse | | | | | | | |
| Blood pressure | | | | | | | |
| Temperature | | | | | | | |
| Respiratory rate | | | | | | | |
| Blood transfusion | | | | | | | |
| IV fluids | | | | | | | |
| RTA | | | | | | | |
| UOP | | | | | | | |
| Drain | | | | | | | |
| Dr ugs | | | | | | | |

17. Postoperative complications:

18. Histopathological report of the specimen:

19. Condition at the time of discharge:

20. Advice on discharge:

21. Follow-up for any complaints:

22. Remarks:

## KEY TO MARTER CHART

| | | |
|---|---|---|
| A | - | absent |
| C | - | cured |
| CA | - | carcinoma |
| D | - | death |
| DIS | - | distension of the abdomen. |
| DVS | - | derotation of the volvulus and sigmoidopexy. |
| F | - | female |
| ICT | - | ileocaecal tuberculosis |
| IP NO. | - | in patient number |
| M | - | male |
| MAFL | - | multiple air – fluid levels |
| Mx | - | mixed diet. |
| ND | - | not done. |
| P | - | present. |
| PR | - | per rectal examination. |
| R | - | rigidity |
| RT | - | rebound tenderness |
| RA | - | resection and anastamosis |
| RA&H | - | resection and anastamosis with herniorraphy. |
| RCJ | - | red currant jelly |
| ROA | - | release of adhesions |
| ROA&H | - | release of adhesions and herniorraphy |
| RTI | - | respiratory tract infections |
| SEP | - | sepsis |
| V | - | vegetarian |
| VIP | - | visible intestinal peristalsis |
| VOM | - | vomiting |
| WNL | - | within normal limits |

# MASTER CHART

| S.NO | NAME | AGE | SEX | IP.NO | DIET | PAIN | VOM | DIS | VIP | TACHYCARDIA | PERITONISM | SCAR | MASS | P/R | DIAGNOSIS | XRAY | TREATMENT | CURED | COMPLICAT | DEATH |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | RAMESH | 17 | M | 1342586 | Mx | P | A | P | P | P | R | P | A | N | ADHESIONS | MAFL | ROA | C | - | |
| 2 | JAYANTHI | 20 | F | 1365746 | V | P | P | P | A | A | RT | A | P | RCJ | INTUSUSCEPTION | ND | REDUCTION | C | - | |
| 3 | KUMARESAN | 66 | M | 1356475 | Mx | P | P | P | P | P | R | P | A | N | ADHESIONS | MAFL | ROA | C | - | |
| 4 | AMBIKA | 23 | M | 1378968 | V | P | P | P | P | P | RT | P | A | N | ADHESIONS | ND | ROA | C | - | |
| 5 | AYYAVU | 55 | M | 1349872 | V | P | P | P | P | P | R | P | A | N | ADHESIONS | MAFL | ROA | C | - | |
| 6 | PERIYACHI | 70 | F | 1357689 | Mx | P | A | A | P | P | RT | A | P | RCJ | INTUSUSCEPTION | ND | REDUCTION | C | - | |
| 7 | PRASANTH | 15 | M | 1378965 | V | P | P | P | P | P | RT | P | A | N | ADHESIONS | MAFL | ROA | C | - | |
| 8 | SUNDARAMBAL | 75 | F | 1379786 | V | A | A | P | P | P | R | A | A | N | CA SIGMOID COLON | MAFL | RA | - | SEP | D |
| 9 | KANDASAMY | 71 | M | 1386754 | Mx | P | P | P | P | P | RT | A | A | N | STRANGULATED ING HERNIA | ND | RA&H | C | - | |
| 10 | PARVATHY | 30 | F | 1389776 | Mx | P | P | A | P | P | RT | A | A | N | OBSTRUCTED ING HERNIA | ND | RA&H | C | - | |

# MASTER CHART

| S.NO | NAME | AGE | SEX | IP.NO | DIET | PAIN | VOM | DIS | VIP | TACHYCARDIA | PERITONISM | SCAR | MASS | P/R | DIAGNOSIS | XRAY | TREATMENT | CURED | COMPLICAT | DEATH |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 11 | RAMASAMY | 72 | M | 1390756 | Mx | P | P | P | P | A | T | A | P | MASS | CA RECTUM | LBO | HARTMANN'S PROCEDURE | | RTI | D |
| 12 | MUZAFFAR | 23 | M | 1392345 | Mx | P | P | P | P | P | R | P | P | N | ADHESIONS | MAFL | ROA | C | - | |
| 13 | SUGANTHI | 26 | F | 1395732 | Mx | A | P | P | P | A | T | A | P | N | ICT | MAFL | RA | C | - | |
| 14 | GANESAN | 85 | M | 1397832 | V | P | P | P | P | P | RT | P | A | N | ADHESIONS | MAFL | ROA | C | - | |
| 15 | ADAIKALAM | 64 | M | 1397865 | Mx | P | P | A | A | P | T | A | A | N | OBSTRUCTED ING HERNIA | ND | RA | C | - | |
| 16 | MUNIYAN | 60 | M | 1399654 | Mx | A | A | P | A | P | T | A | A | N | OBSTRUCTED ING HERNIA | ND | ROA&H | C | - | |
| 17 | SENTHIL | 25 | M | 1399976 | V | A | P | P | P | P | RT | P | A | N | ADHESIONS | MAFL | ROA | C | - | |
| 18 | KANNAMMAL | 70 | F | 1412367 | V | P | P | P | A | P | RT | A | A | RCJ | SIGMOID VOLVULUS | ND | DVS | C | - | |
| 19 | JANAKI | 50 | F | 1416783 | Mx | P | A | P | A | P | RT | A | A | N | STRANGULATED ING HERNIA | ND | RA | C | - | |
| 20 | RAGUPATHY | 60 | M | 1427568 | Mx | P | P | A | P | P | R | A | A | N | OBSTRUCTED HERNIA | ND | ROA&H | C | - | |

# MASTER CHART

| S.NO | NAME | AGE | SEX | IP.NO | DIET | PAIN | VOM | DIS | VIP | TACHYCARDIA | PERITONISM | SCAR | MASS | P/R | DIAGNOSIS | XRAY | TREATMENT | CURED | COMPLICAT | DEATH |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 21 | JOTHILINGAM | 65 | M | 1432534 | Mx | P | P | P | A | P | T | A | A | N | MESENTERIC ISCHEMIA | MAFL | RA | | SEP | D |
| 22 | GANESAN | 45 | M | 1436574 | V | P | A | P | A | P | R | A | A | N | SIGMOID VOLVULUS | MAFL | DVS | C | - | |
| 23 | KANDAN | 40 | M | 1436787 | Mx | P | P | P | P | A | R | P | A | N | ADHESIONS | MAFL | ROA | C | - | |
| 24 | RAMAN | 40 | M | 1430897 | V | P | P | P | A | P | R | A | A | N | OBSTRUCTED ING. HERNIA | ND | ROA&H | C | - | |
| 25 | MURUGESAN | 45 | M | 1445674 | Mx | P | P | P | P | P | RT | A | A | N | STRENGULATED ING. HERNIA | ND | RA | C | - | |
| 26 | MOORTHY | 60 | M | 1449687 | Mx | P | P | P | P | A | T | P | A | N | ADHESION | MAFL | ROA | C | - | |
| 27 | KRISHNAN | 35 | M | 1449807 | Mx | P | P | P | P | P | RT | P | A | N | ADHESION | MAFL | ROA | C | - | |
| 28 | PECHAYEE | 69 | F | 1451295 | V | P | P | P | A | A | RT | A | P | RCJ | INTUSUSCEPTION | - | RA | C | - | |
| 29 | KANAGA | 40 | F | 1452313 | Mx | P | P | P | P | P | RT | P | A | N | ADHESION | MAFL | ROA | C | - | |
| 30 | SUNDARAM | 29 | M | 1453271 | Mx | P | P | P | P | A | T | P | A | N | ADHESION | MAFL | ROA | C | - | |

# MASTER CHART

| S.NO | NAME | AGE | SEX | IP.NO | DIET | PAIN | VOM | DIS | VIP | TACHYCARDIA | PERITONISM | SCAR | MASS | P/R | DIAGNOSIS | XRAY | TREATMENT | CURED | COMPLICAT | DEATH |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 31 | SURENDAR | 18 | M | 1455637 | Mx | P | P | P | P | P | T | A | A | N | OBSTRUCTED ING. HERNIA | - | ROA&H | C | - | |
| 32 | SELVI | 25 | F | 1456374 | Mx | P | A | P | P | P | T | P | A | N | ADHESIONS | MAFL | ROA | C | - | |
| 33 | RAMU | 35 | M | 1456893 | V | P | P | P | P | A | RT | P | A | N | OBSTRUCTED ING. HERNIA | ND | ROA&H | C | - | |
| 34 | RATHINAM | 65 | M | 1458965 | Mx | P | P | P | A | A | R | P | A | N | OBSTRUCTED ING. HERNIA | ND | ROA&H | C | - | |
| 35 | SURESH | 18 | M | 1463214 | Mx | P | A | P | A | P | RT | P | P | N | OBSTRUCTED ING. HERNIA | ND | ROA&H | C | - | |
| 36 | RAVIKUMAL | 45 | M | 1468432 | Mx | A | A | P | P | A | T | A | P | N | CA CECUM | MAFL | PA | | RTI | D |
| 37 | SHANTHI | 38 | F | 1469354 | Mx | P | P | P | P | P | RT | A | P | N | CA OVARY ADHESION WITH SIGMOID COLON | MAFL | TLC | | SEP | D |
| 38 | AROKIASAMY | 35 | M | 1469756 | V | P | P | P | P | P | T | P | A | N | ADHESIONS | MAFL | ROA | C | - | |
| 39 | RAJENDRAN | 63 | M | 1461982 | V | A | P | P | A | P | R | A | A | MASS | CA RECTUM | MAFL | HARTMANN'S PROCEDURE | | SEP | D |
| 40 | KALAISELVI | 25 | F | 1461993 | Mx | P | P | P | A | P | RT | P | A | N | ADHESIONS | MAFL | ROA | C | | |

# MASTER CHART

| S.NO | NAME | AGE | SEX | IP.NO | DIET | PAIN | VOM | DIS | VIP | TACHYCARDIA | PERITONISM | SCAR | MASS | P/R | DIAGNOSIS | XRAY | TREATMENT | CURED | COMPLICAT | DEATH |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 41 | RAMANUJAM | 38 | M | 1463562 | Mx | P | P | P | A | P | T | P | A | N | OBSTRUCTED ING. HERNIA | ND | ROA&H | C | | |
| 42 | DAVID | 40 | M | 1465342 | Mx | P | P | P | P | A | RT | A | A | N | ADHESIONS | MAFL | ROA | C | | |
| 43 | RANGASAMY | 55 | M | 1465372 | V | P | P | P | P | P | RT | A | P | N | CA COLON | MAFL | RA | | SEP | D |
| 44 | KUMARAN | 55 | M | 1475632 | Mx | P | P | P | A | P | RT | A | A | N | OBSTRUCTED ING. HERNIA | ND | RA&H | C | | |
| 45 | RAMESH | 20 | M | 1477563 | V | P | P | P | A | P | R | A | P | N | OBSTRUCTED ING. HERNIA | ND | ROA&H | C | | |
| 46 | MUTHAMMAL | 55 | F | 1479563 | V | P | A | P | P | P | T | P | A | N | ADHESIONS | MAFL | ROA | C | | |
| 47 | SAROJA | 40 | F | 1483213 | Mx | P | P | A | P | A | RT | P | P | N | STRANGULATED ING. HERNIA | MAFL | RA&H | C | | |
| 48 | KANNAPAN | 52 | M | 1487632 | Mx | P | P | P | P | P | R | P | A | N | ADHESIONS | MAFL | ROA | C | | |
| 49 | MUTHUSAMY | 60 | M | 1487653 | Mx | P | P | P | P | P | RT | A | P | N | ICT | MAFL | RA | C | | |
| 50 | RAMAYEE | 58 | F | 1489032 | Mx | P | A | P | P | P | RT | A | A | N | ADHESIONS | MAFL | ROA | C | | |